EVALUATION OF C- REACTIVE PROTEIN (CRP) IN NEONATAL SEPSIS IN COMPARISON WITH CELLULAR AND CLINICAL PARAMETERS

Dissertation Submitted to

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CHENNAI, TAMILNADU

APRIL 2018

CERTIFICATE

This is to certify that the dissertation titled, *"***Evaluation of C - Reactive Protein (CRP) in neonatal sepsis in comparison with Cellular and Clinical parameters***"* is a genuine work done by **Dr. V.Gomathi**, for the partial fulfillment of the requirements for M.D (Biochemistry) Branch XIII Examination of The Tamil Nadu Dr. M.G.R. Medical University to be held in April 2018 during the academic period 2015-2018.

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This is to certify that the dissertation on **"Evaluation of C-Reactive Protein (CRP) in neonatal sepsis in Comparison with Cellular and Clinical parameters"** is a record of research work done by **Dr.V.Gomathi** in partial fulfilment for M.D (BIOCHEMISTRY) Examination of The Tamil Nadu Dr. M.G.R. Medical University to be held in April 2018.The period of study is from February 2017 to July2017

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DECLARATION

I, **Dr. V. Gomathi**, solemnly declare that the dissertation titled **"Evaluation of C- Reactive Protein (CRP) in neonatal sepsis in Comparison with Cellular and Clinical parameters"** is a bonafide work done by me during the period of FEBRUARY 2017 to JULY 2017 at Government Stanley Medical College and Hospital, Chennai under the expert guidance of

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ABBREVIATIONS

CRP –C-Reactive Protein

I/T -Immature granulocyte to Total neutrophil ratio

ANC- Absolute Neutrophil Count

TLC-Total Leukocyte Count

WBC-white blood cell

WHO-World Health Organisation

EOS –Early onset of sepsis

LOS-late onset of sepsis

PROM-Premature Rupture of Membrane

MSAF- Meconium Stained Amniotic Fluid

CRT-Capillary Refilling Time

IG –Immature Granulocyte

LBW-Low Birth Weight

VLBW-Very Low Birth Weight

PPV-Positive Predictive Value

NPV-Negative Predictive Value

INDEX

INTRODUCTION

Neonatal sepsis is a systemic infection that occurs within 4 weeks of life[1]. Neonatal sepsis is one of the foremost cause of neonatal mortality and morbidity worldwide. Neonatal sepsis is classified into early onset sepsis (EOS) in which the symptoms occurs within first 72 hours of life and late onset sepsis (LOS) in which the symptoms occurs after 72 hours of life.[2]The incidence of neonatal sepsis still remains high inspite of the improvement in the perinatal and neonatal care. In developed countries, the sepsis rates range from 10-25% for all neonates to around 50% in preterm very low birth weight (VLBW) infants[3,4.].The figures in the developing countries are still considerably higher even though the exact figure is not known. World Health Organization (WHO) estimates that out of the four million neonatal deaths all over the world every year, over 35% are related to infection in the neonatal period[5,6,7]. In India, the incidence of neonatal sepsis varies from 11-24.5/1000 live births and about 1-8 cases in all livebirths.[8] In developing countries 30 -50% of neonatal death in each year is probably due to neonatal sepsis.[9,10]

The preterm and low birth weight babies are more prone to sepsis than term babies. The reasons are mainly due to the combination of the neonatal reduced immune defense and the complex interactions between the infecting microorganisms and the host response[10,11].Early clinical presentation of sepsis is non- specific, vague, ill-defined and difficult to

differentiate infectious condition from non-infectious condition. For neonates, progression of infection is more rapid than adults and cause serious complications[7]. Early diagnosis of disease is therefore very important for treatment and prevention of further complications.

Many investigations are done to diagnose sepsis. A best diagnostic test should have excellent sensitivity, negative predictive value (NPV) as well as excellent specificity and Positive Predictive Value. It should be cost effective and yield results early[12,13].Gold standard diagnostic test for neonatal sepsis is blood culture which is time consuming, has low positivity, limited availability and also the results are available after 48 hrs -72 hrs [14]. Many investigators have evaluated new markers like cytokines, cell surface antigens, procalcitonin for rapid diagnosis of sepsis, but their use in routine practice are limited by the lack of resources in rural areas and high cost which are impractical for the developing countries.[13,14,16,17]

Despite advanced bacteriologic techniques, different investigative techniques were assessed for the early prediction of neonatal sepsis. Much work has been done on assessing the role of various parameters like C reactive protein (CRP), hematological parameters(Cellular parameters) such as Total Leukocyte Count (TLC), Absolute Neutrophil Count (ANC), Immature granulocyte : Total neutrophil ratio (I/T),platelet count in diagnosing neonatal sepsis.These tests are less expensive than the new sophisticated markers and blood culture. The results could be obtained

earlier than the blood culture and are useful in early initiation of antibiotic treatment, which is helpful to reduce neonatal mortality and morbidity.[18]

These hematological markers and c-reactive protein combined with clinical assessment, increase the probability of correct diagnosis and offer paediatrician greater confidence in promptly initiating antimicrobial therapy, in parallel with supportive care. On the other hand, they can also avoid the indiscriminate use of antibiotic treatment, which reduces the risk of developing multi-resistant pathogens and help to reduce hospital costs.

REVIEW OF LITERATURE

NEONATAL SEPSIS

DEFINITION

Neonatal sepsis refers to systemic infection affecting infants with in 1month of life and is typically of bacterial origin. It basically includes invasion of blood stream by pathogens and most of the time may involve multiple organ system.

The term neonatal sepsis covers blood stream infections (BSIs) or Septicemia . It also covers meningitis, urinary tract infection and joint/bone infection but does not include superficial infections[19]

CLASSIFICATION:

A.Based on age of onset of symptoms neonatal sepsis is classified into

1.EARLY ONSET OF SEPSIS (EOS)

Sepsis occurs within 72 hours of birth

2.LATE ONSET OF SEPSIS (LOS)

Sepsis occurs at or beyond 72 hours of birth

B. Based On Culture (The national neonatology forum's definition of sepsis for hospitals)[20]neonatal Sepsis is Classified into

1.Confirmed sepsis(Culture positive)

2.Clinical sepsis(Culture negative)

CONFIRMED OR CULTURE POSITIVE SEPSIS:

Infants with clinical symptoms and signs of sepsis with isolation of pathogen from blood ,urine & CSF culture

CLINICAL OR CULTURE NEGATIVE SEPSIS:

Infants with clinical signs and symptoms of sepsis with culture negative but associated with one or more of the following criteria

1.PREDISPOSING FACTORS: Maternal fever or foul smelling liquor or Pronloged rupture of membranes >12 hours or presence of gastric polymorphs

2**.POSITIVE SEPSIS SCREEN**

TLC<5000/mm^3

I/T>0.2

CRP>0.6µg/ml

ESR>10mm1st hour

3**.RADIOLOGICAL**evidence of pneumonia[20]

EPIDEMIOLOGY

Globally, sepsis is one of the major cause of neonatal mortality and morbidity, inspite of new advances in health care units[21]. Out of 5million neonatal deaths per annum, from which 30-40% of deaths are due to infections[22].

Incidence of neonatal sepsis in developed countries ranges from 1to 4 / 1000 live Births[23] .In India the incidence of neonatal sepsis is 30 / 1000 live births (according to the data from National Neonatal Perinatal Database[24] (NNPD, 2002-03) which is 3-20 times higher than the incidence rate of developed Countries[19].

The database encompassing 18 tertiary care neonatal units across India found sepsis as one of the commonest causes of neonatal mortality contributing to 19% of all neonatal deaths[23].Neonatal sepsis is most common in low birth weight infants and early gestational age. It is 10 times more commoner in very low birth weight neonates (VLBW infants – birth weight <1500g). In developing countries VLBW infant's septicemia ranges between 20 –41%.[19]

Incidence of central vein associated blood stream infection (CLABSI) is 12.2% and ventilator associated pneumonia (VAP) is 9/1000 . Globally Neonatal infections kill nearly 11% of children under five years of age group ,in which India contributes major fraction of nearly 40% .This percentage of septic infants have long term sequelae in which the most common are developmental delay (74%),cerebral palsy (36%), visual impairment (32%) and hearing impairment (10%).The risk increases further in LBW and small for gestational age neonates but remains similar in EOS or LOS[19].

ETIOLOGY

Major pathogens causing neonatal sepsis detected in developing countries and developed countries are listed in the table.no(1.) Microorganisms causing EOS and LOS are largely identical in developing countries but in developed countries these pattern varies where group B streptocooci and E.coli dominating EOS.

PATHOGEN PROFILE IN NEONATAL SEPSIS[25]

Developing countries	Developed countries
1.klebsiella pneumonia 2.Acinetobacter spp 3.Escherichia coli 4.psedomonas spp 5.Staphylococcus aureus	1.Group B streptococci 2.Escherichia coli 3.Coagilus negative staphylococci 4.Staphylococcus aureus 5.Enterococcus spp 6.Listeria monocytogens

Table 1 –shows pathogen profile in neonatal sepsis

Mode of transmission[19]

The new born infant can acquire infection basically by three ways. They are

1.Trans placental route (Manifest as congenital infection e .g rubella, toxoplasma and listeria etc)

(OR)

2.Vertical infections from mother during the process of labour and delivery (e.g group B Streptococcus and gram negative infection)

(OR)

3.Horizontal infection from environment or care takers after delivery (e.g Coagulase +ve and Coagulase – ve staphylococcus)

RISK FACTORS OF SEPSIS[19]

Risk factors are broadly divided into three types

1. Maternal risk factors
2. Neonatal risk factors
3. Environmental factors

1. MATERNAL RISK FACTORS ARE:

a. Fever
b. Bacteriuria (symptomatic)
c . Maternal amnionitis
c. Prolonged rupture of membrane
e . Excessive bleeding
f . Colonization of organism in genitourinary tract
g. Socio economic factors (poor hygiene and poor nutrition, higher incidence of LBW and thereby causes higher rate of infection)

2. NEONATAL RISK FACTORS ARE[19]

a. Preterm
b. Lower birth weight(LBW)
c. Male gender
d. Twins (1^{st} born)
e. Congenital malformation associated with interruption of mucosa and skin
f. Exposure to particular drugs (e.g steroids)
g. Deficiency of humoral and cellular immunity –mainly in preterm

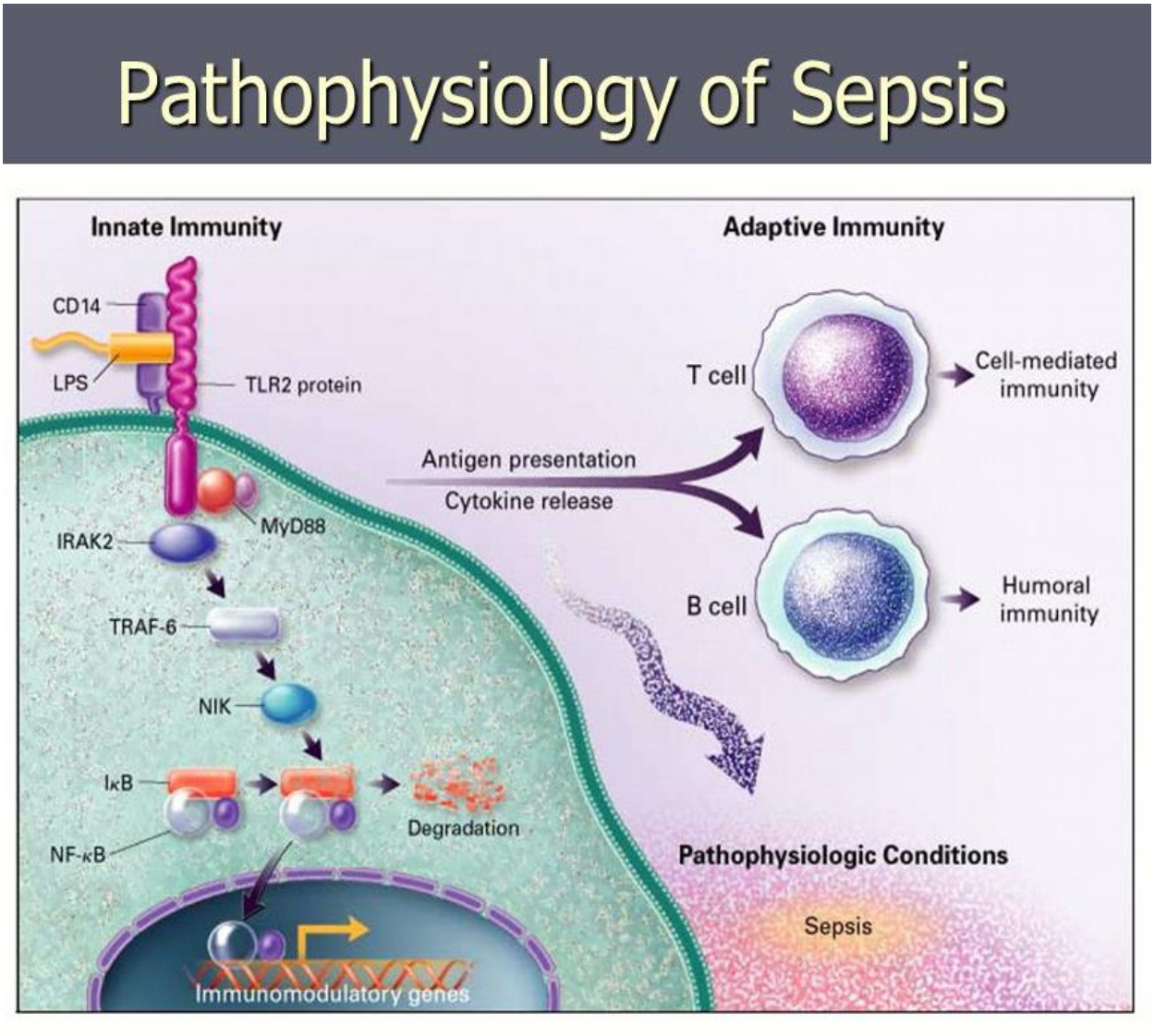
3 .ENVIRONMENTAL FACTORS[19]

a. Aspiration of contaminated amniotic liquor viz fetal distress, asphyxia
b. Flora present in hospital and home , caretakers and equipments
c. Invasive procedure and resuscitation which leads to bacterial infection.

PATHOPHYSIOLOGY OF NEONATAL SEPSIS

Sepsis is generally considered as unusual systemic response to an ordinary infection. Manifestation of sepsis results from interaction between host, pathogen and environement. Host defense against invading pathogens mainly depend on the innate and adaptive immunity .

Figure 1. pathophysiology of sepsis[26]



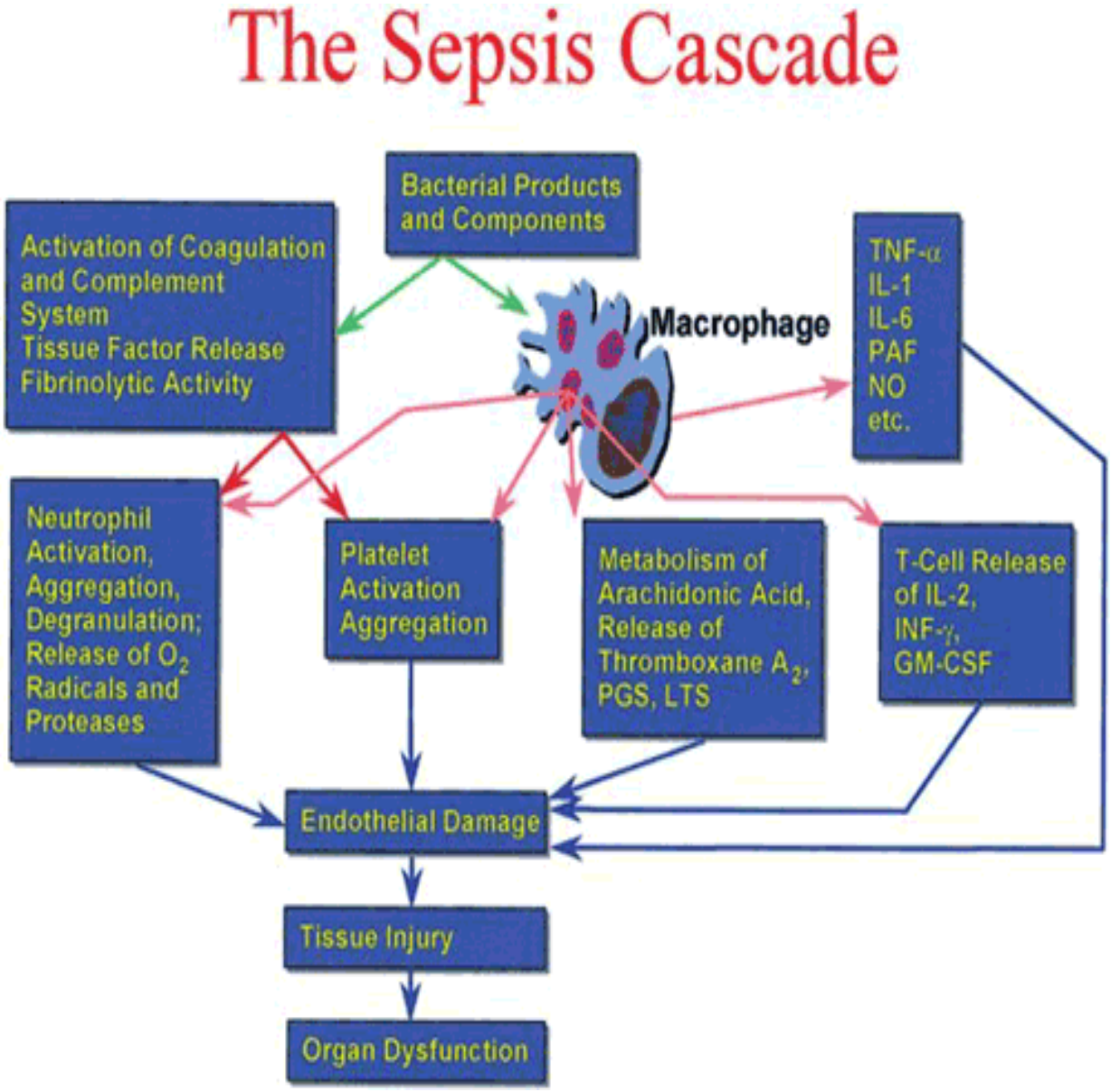


Figure 2: Sepsis cascade[26]

INNATE IMMUNE SYSTEM

It consists of phagocytes,complement system and antigen presenting cells (monocytes, macrophage ,polymorpho nuclear cells and dentritic cells)[27]

Once pathogen enters into the local tissue PAMPs (Pathogen associated molecular patterns) is released .PAMPs of gram positive bacteria is LTA (Lipoteichoic acid) and gram negative bacteria is LPS (Lipo polysaccharide). Then PAMPs binds to immune cell in host through pathogen recognition receptors (PRRs).At the same time host cell releases damage associated molecular patterns (DAMPs)[19]

DAMPs together with PAMPs stimulate innate immune response (chiefly tissue macrophage and complement system) leading to the release of pro-inflammatory markers like cytokines(IL-6,TNF-α,IL-1β) and chemokines (IL-8) resulting in clinical symptoms and signs of sepsis[19]When pathogen enters into blood stream. It causes systemic reactions via pro inflammatory response (Systemic inflammatory response syndrome (SIRS)).

Immune system overboard to destroy the spread of infection or neutralise the attack by producing pro inflammatory markers which may cause harm to its own tissues and organs leading to multi organ failure[19]

LIMITATION ON INNATE IMMUNE SYSTEM IN NEWBORN

Neonates innate immune system have less capacity to produce cytokines like IL6 and TNF-α and simultaneously produce anti-inflammatory cytokines IL-10 which inhibit the synthesis of pro - inflammatory cytokines.

Host immune system produce compensatory anti- inflammatory cytokines. But immature production of anti- inflammatory cytokines reported in both preterm and term infants may be associated with multiorgan dysfunction (MOD) and shock –Identified clinically as severe sepsis.[19]

Neutrophils, nature killer cells and dendritic cells are less in number and functionally impaired in neonates.The neonates complement system possess half of adults level even at term gestation reflect defective opsonisation and defective bacterial killing.

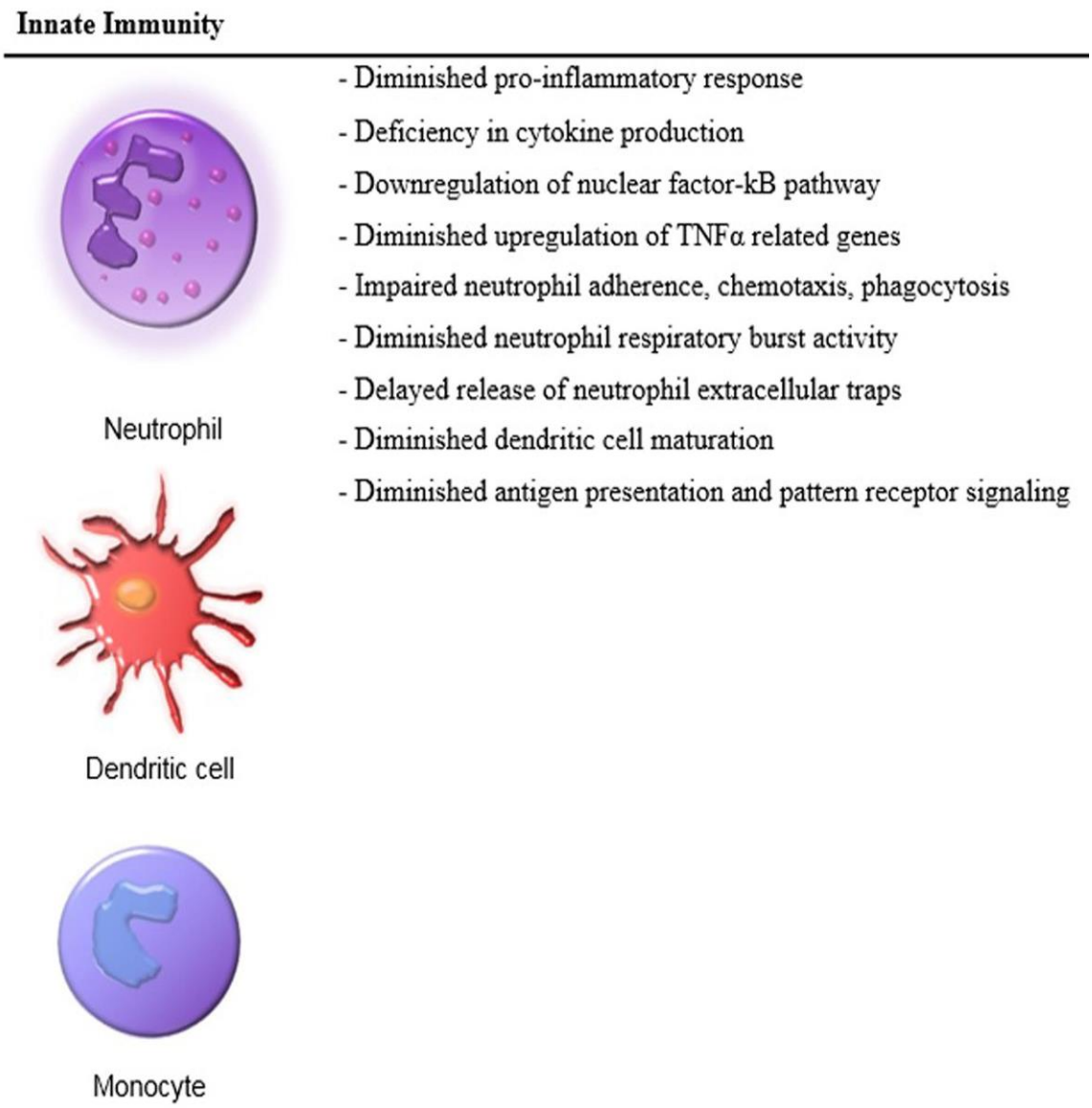
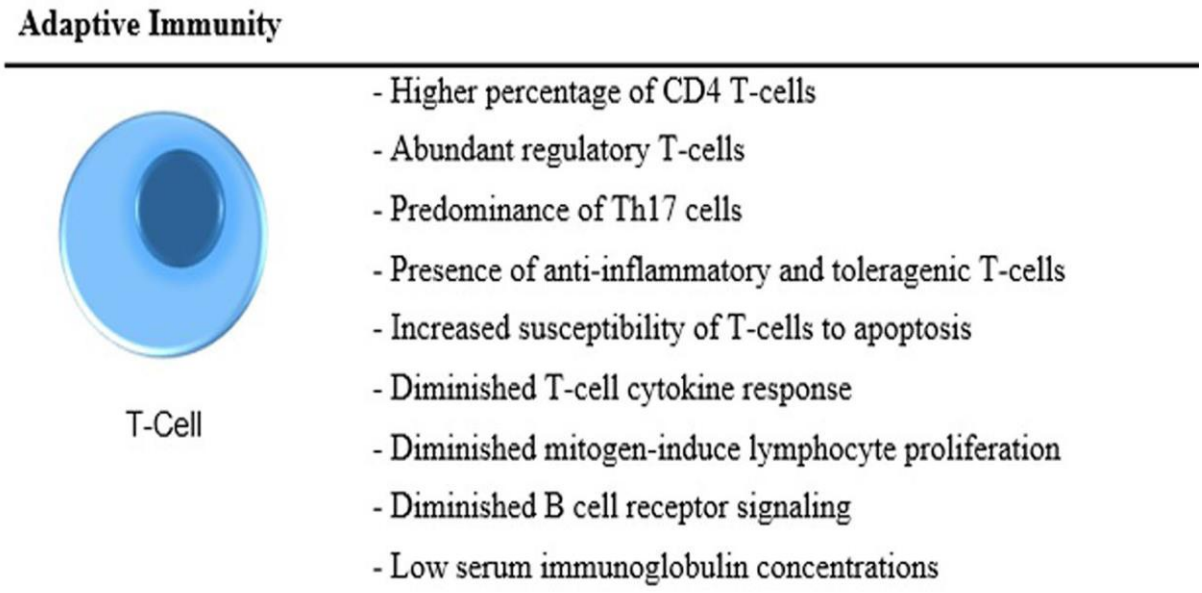


FIGURE 3 –LIMITATION OF INNATE IMMUNITY IN NEWBORN[27]

LIMITATION OF ADAPTIVE IMMUNE SYSTEM IN NEONATES

Neonates have weak pro inflammatory immune reponse .This leads to decreased cytotoxic function of B cell and decreased isotype switching which leads to defective cell mediated immunity.so following primary infection, a delay occurs in formation of antigenic specific memory function which can worsens this problem. Even at term neonates having low immunoglobulin concentration which is responsible for decreased adaptive immunity.[19]

FIGURE -4 SHOWS LIMITATION OF ADAPTIVE IMMUNITY[27]



PATHOGENESIS OF EARLY ONSET OF SEPSIS

Usually before birth, the fetus is optimally maintained in a sterile environment.In Early onset of sepsis infection transmitted vertically from mother to fetus through trans placental or ascending infection from genito urinary tract.

Organism causing EOS may ascend from birth canal due to rupture of amniotic membrane or leak before labour or during process of the labour causing intra-amniotic infection[28] .Infection of the amniotic fluid, membranes,and placenta, and/or decidua) together is referred to as "chorioamnionitis'. Chorioamnionitis is a one of the main risk factor for neonatal sepsis. Sepsis can occur in utero when the fetus inhales or swallows infected amniotic fluid. The neonate can also develop sepsis in the hours or days after birth due to colonized skin or mucosal surfaces .. Maternal fever is the essential criteria for the clinical diagnosis of chorioamnionitis.

Most common pathogens causing EOS are – Group B streptococcus, E.coli, H.influenzae and Listeria monocytogens. Most Common organism causing EOS in India are Escherichia coli ,klebsiella pnemoniae and S.aureus.

The diagnosis of chorioamnionitis is typically based on

1. The presence of maternal fever (greater than 38°C (100.4°F)) and at least two of the following criteria:
    a. Maternal leukocytosis (greater than 15 000 cells/mm3)
    b. Maternal tachycardia (greater than 100 beats/minute)
    c. Fetal tachycardia(greater than 160 beats/minute)
    d. Uterine tenderness, and/or foul odour of the amniotic fluid.[29]

These findings contribute higher rates of neonatal and maternal mortality. Approximately 14% to 28% of women delivering preterm babies at 22 to28 weeks of gestation exhibited signs compatible with chorioamnionitis.[30]

The major risk factors for chorioamnionitis are listed below

1. Low parity,
2. Spontaneous labour
3. Longer length of labour and membrane rupture,
4. Multiple digital vaginal examinations (especially with ruptured membranes),
5. Meconium-stained amniotic fluid, and presence of genital tract microorganism (e.g Mycoplasma hominis).[31]

The major risk factors for early-onset neonatal sepsis are

1. Pre -term birth

2 . Maternal colonization with GBS

3 . Rupture of membranes >18 hours, and Maternal signs or symptoms of intra-amniotic infection[32,33,34].

Other variables include

a. Ethnicity (black women are at high risk of being colonized with GBS)

b. low socioeconomic status, male sex

c .Low Apgar scores

d .Low birth weight

The increased risk of early-onset sepsis in preterm infants are due to complications of labour and delivery and poor development of innate and adaptive immunity.[35]

PATHOGENSIS OF LATE ONSET OF SEPSIS(LOS)

LOS is most frequently defined as appearance of symptoms after 72 hrs of birth.LOS is acquired through postnatal nosocomial infections and care giving practices in home or community environment. The peak incidence seen in between the 10th and 22nd day of life[36,37,38, 39].

The incidence of LOS is inversely proportional to birth weight (BW). Likewise,36.3% of neonates with gestational age (GA) <28 weeks had at least one episode of LOS, as compared with 29.6%, 17.5% and 16.5% of

moderately preterm (GA of 29–32 weeks), late preterm (GA of 33–36 weeks) and term infants respectively[38]

RISK FACTORS OF LATE ONSET OF SEPSIS[19]

a. LBW (<2500g)
b. Preterm (<37 weeks)
c. Admission in NICU
d. Administration of IV fluids
e. Mechanical ventilation
f. Invasive procedure
g. Presence of central lines
h. Unnecessary investigation
i. Poor hygiene/poor cord care
j. Bottle feeding

Common pathogens causing Late onset of sepsis[19]

1. Klebsiella
2. Staph aureus
3. E.coli
4. Enterococci
5. Pseudomonas
6. Anaerobes

Table 2- shows differences in early and late onset of sepsis[19]

	EOS	LOS
1.Time of onset	<72hours	>72hours
2. Due to complication of pregnancy or delivery	+	+ or -
3.Source of organism	Mother's genital tract	Nosocomial and mothers genital tract
4.clinical presentation	Fulminant /multisystem involved/pneumonia	Slowly progress, Mostly no pnemonia .
5.Mortality	30-50%	15-30%
6.common microorganism	Klebsiella, Listeria GBS(rare in India)	Staph aureus Klebsiella E.coli Enterocooci Pseudomonas anarobes
7.Meningitis	Occurs rarely	occurs commonly

CLINICAL FEATURES OF NEONATAL SEPSIS

The earliest signs of sepsis are often asymptomatic and nonspecific indeed, high index of suspicion is needed for early diagnosis. This is more true in preterm babies.[39]

NONSPECIFIC FEATURES OF SEPSIS

1. Hypothermia or fever (hypothermia more common in preterm low birth weight Infants)
2. Lethargy ,poor cry ,refusal to suck
3. Poor perfusion and prolonged capillary refilling time
4. Hypotonia ,absent neonatal reflex
5. Respiratory distress ,apnea ,and gasping
6. Bradycardia /tachycardia
7. Hypo and hyperglycemia and metabolic acidosis

SPECFIC FEATURES ASSOCIATED WITH VARIOUS SYSTEMS

CENTRAL NERVOUS SYSTEM (CNS)

Bulging anterior fontanelle, excess irritability ,high pitched cry, stupor ,coma, seizures, neck retractions presence of above features raise the clinical suspicion of meningitis.

CARDIOVASCULAR SYSTEM

1. Hypertension
2. Poor perfusion
3. Shock

RESPIRATORY SYTEM

1. Apnea

2. Cyanosis

3. Tachypnea

4. Chest retraction

5. Grunting

GASTRO INTESTINAL SYSTEM

Feeding intolerance, vomiting, diarrhea, abdominal distension ,paralytic ileus, necrotizing enterocolitis

HEPATIC

Hepatomegaly, direct hyperbilirubinemia

RENAL

Acute renal failure

HAEMATOLOGICAL

1. Bleeding

2. petechiae

3. purpura

SKIN CHANGES

Abscess, sclerema, mottling ,multiple pustules, umblical redness and discharge

TABLE 3 -MOST FREQUENT SIGNS AND SYMPTOMES OF NEONATAL SEPSIS[27,40]

SYMPTOMES	SIGNS
Lethargy	Temperature instability(>37c° or <36.5°c)
Poor feeding	Prolonged capillary refilling time(>3 sec)
Apnea	Heart rate(>180/min or <100/min)
Respiratorydistress	Does not look well
(Respiratoryrate	Widening of toe- core temperature
>60,grunting.severechest	Hepatomegaly
indrawing,cyanosis)	Splenomegaly
Mottling	Abnormal neurological reflex
Cyanosis	Glucose intolerance
Pallor	a.Hypoglycemia
Abdominal distension	b.Glucosuria
Vomiting	Persistent acidosis
Jaundice	Multiple skin pustules (>10)
Purpura,petechiae,bleeding	Painful bones/joints
Irritability	Sclerema
Seizures	Bulging fontanelle
Diarrhea	
Umbilical discharge	

INVESTIGATIONS FOR SEPIS

1.Direct method:

Body fluid culture (Blood, CSF, urine) -Gold standard test

2.Indirect method:

Early diagnosis of sepsis depends on some indirect markers of infection.

Indirect cellular markers:

Total leucocyte count

Absolute neutrophil count

Immature to total neutrophil ratio (I/T)

ESR

Platelet count

Indirect biochemical markers:

A)ACUTE PHASE REACTANTS

a. CRP
b. Serum haptoglobin
c. Serum oscomucoid
d. Fibronectin
e. TNFα

B).NEWER MARKERS

IL-6, IL-8

PCT (procalcitonin)

CD11b

CD 64

OTHER ASSOCIATED INVESTIGATION

1. Lumbar puncture (In case of suspected meningitis)

2. Chest x ray (Considered in case of respiratory problems)

3. Abdominal x ray (Indicated in abdominal signs suggestive of sepsis)

4. Study of leucocyte in gastric aspirate

C-REACTIVE PROTEIN

CRP is an acute phase protein secreted from the liver in response to any inflammation and play a main role in innate immunity .CRP level rises to peak after 6hrs of acute phase response . CRP has short half life of around 18-19 hrs and so the level decreases fast once the source is removed[41]

HISTORY AND NOMENCLATURE

CRP is an acute phase protein first described by Tillet and Francis in 1930 at Rockefeller University[42]. They observed a substance which was present in sera of acutely ill patients which reacted with C-polysaccharide of Sterptococcus pneumonia causing agglutination[43].In 1941 the substance was identified as a protein, so named C-reactive protein (CRP)[44] .

GENETIC LOCATION AND STRUCTURE

The location of CRP gene is on chromosome - 1 (1q23.2)[45]. CRP consists of five non glycosylated polypeptide subunits which are identical and has a molecular weight of 23028 Da ,non -covalently link to form disc like structure with radial symmetry and total mass about roughly 115 K Da[44]. CRP is belong to pentraxin family and has 224 amino acids.[46]

STRUCTURE OF C-REACTIVE PROTEIN (CRP)

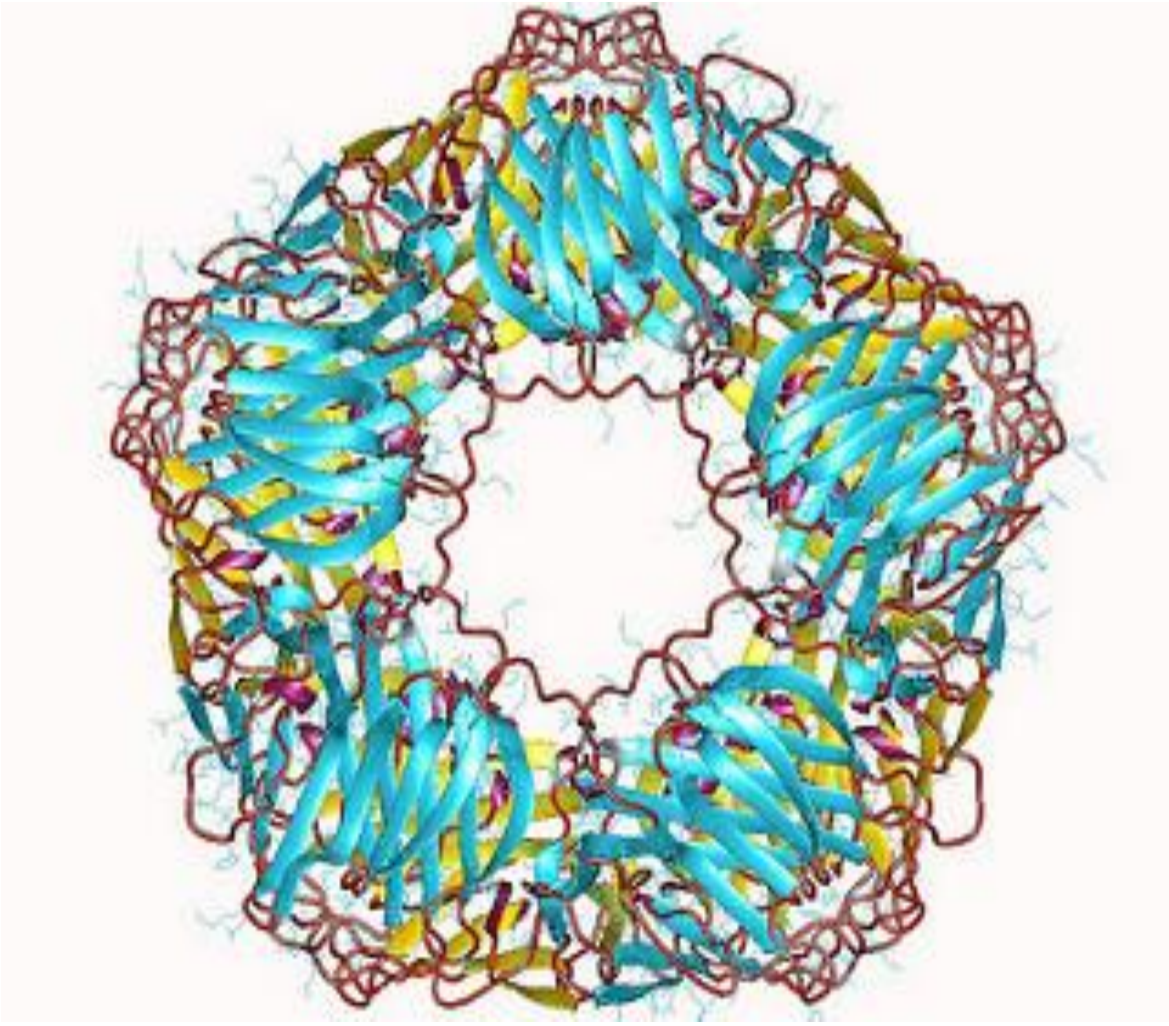


Figure 3:Structure of CRP protein[47]

BIOCHEMICA L FUNCTION[48]

In the presence of Calcium, CRP protein binds not only the C-polysaccharides present in many bacteria, fungi, and protozoa, parasites but also bind with

(a) phosphorylcholine;

(b) phophatidylcholines, like, lecithin; and

(c) polyanions, such as nucleicacids.

In the absence of Ca^{2+}, CRP binds polycations such as histones.

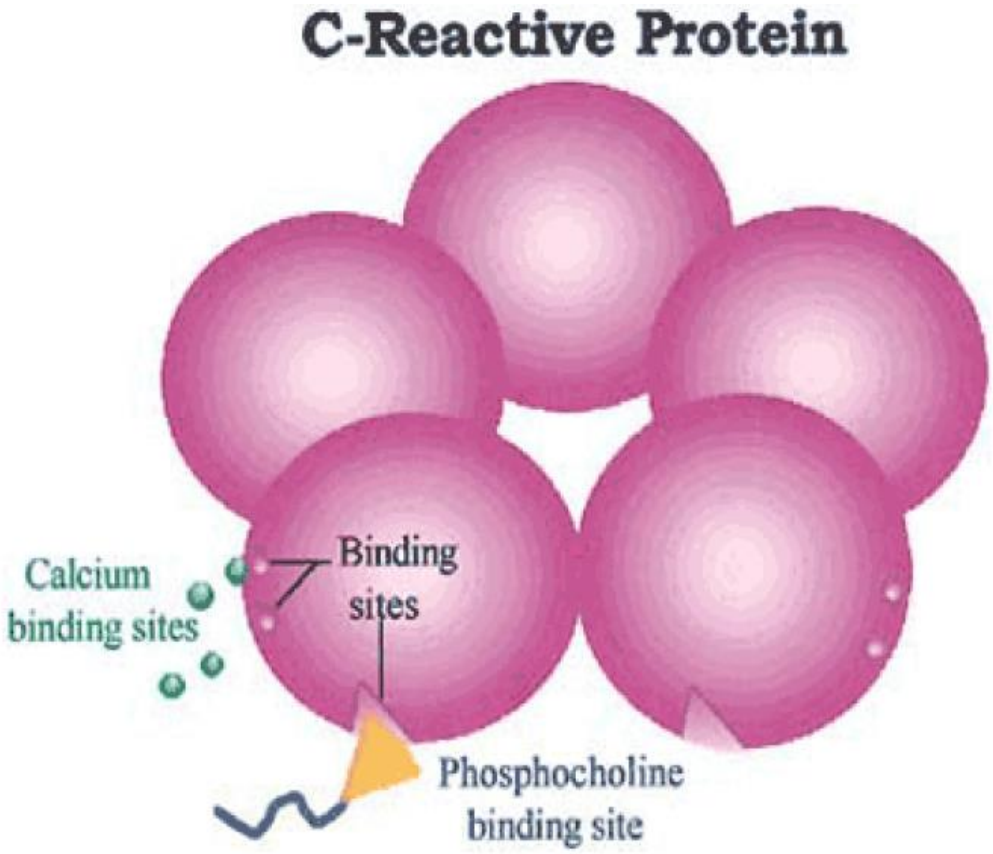


Figure 4:calcium and phosphocholine binding site of CRP[47]

This complexed CRP activates classical complement pathway and initiate opsonization, phagocytosis, destroy the invading pathogens like bacteria and virus .CRP ligand complex also bind to polymorphonuclear leukocytes (PMNs) and monocytes, and stimulate the release of Inflammatory cytokines.[49]

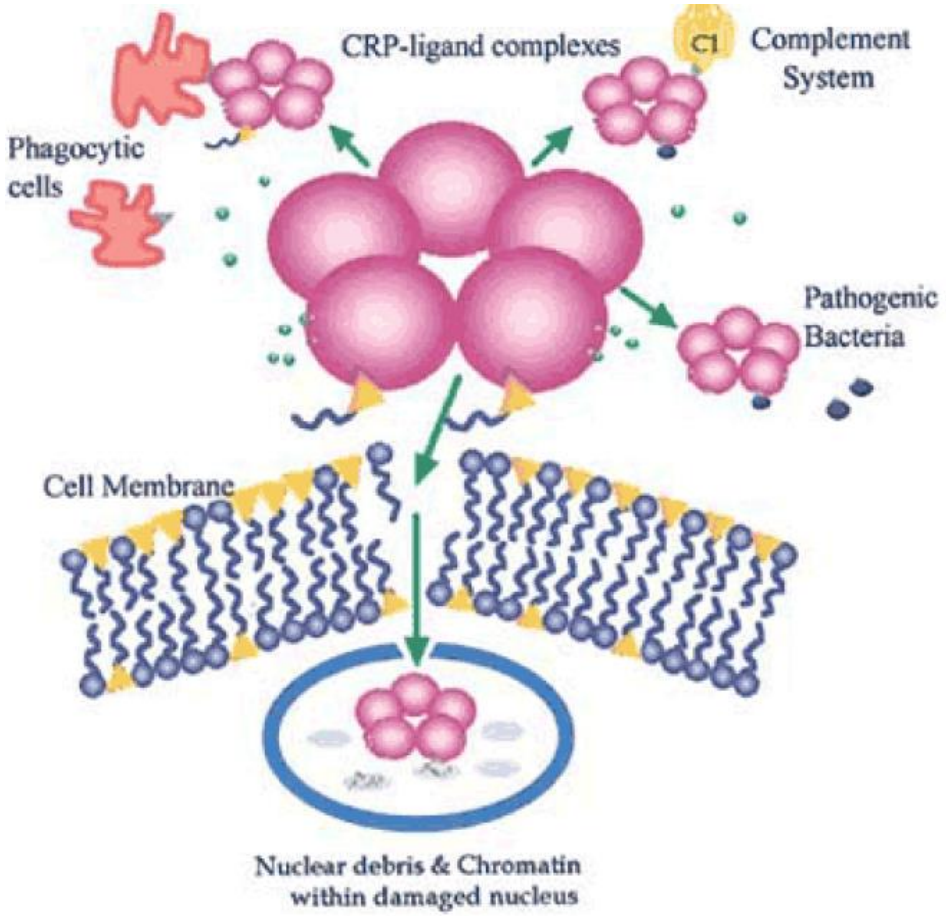


Figure :5 Activation complement system by CRP[47]

It does this function in a manner analogous to that seen with antibody-antigen complexes. It can also recognize potentially toxic substances which are released from damaged tissue, bind with them, and then detoxify toxic substances or clear them from the blood. Half life of

CRP - is 18 to 20 hrs. Then CRP itself should be catabolized after opsonisation So complement activation by CRP binding is one of its role in Innate Immunity (as an early defence system against infections) and may be important in early response to infection in immunosuppressed infants before high concentration of antibody production against infection[50]

CLINICAL SIGNIFIGANCE OF CRP

ACUTE PHASE RESPONSE AND CRP

Acute inflammatory response is a systemic response occurs due to tissue injury caused by any infections or non infections agents like physical, chemical and Immunological toxins.

CRP is one of the positive acute phase proteins the level of which is increased during acute phase response and is usually regulated by cytokines.

In our body during infection, inflammation and tissue injury, circulatory Inflammation mediated cytokines (IL6,TNF,and IL-1) are stimulated which further stimulate the synthesis and release of CRP and other positive acutephase proteins in serum like amyloid A, haptoglobin, and fibrinogen which are released from liver.[51,52]

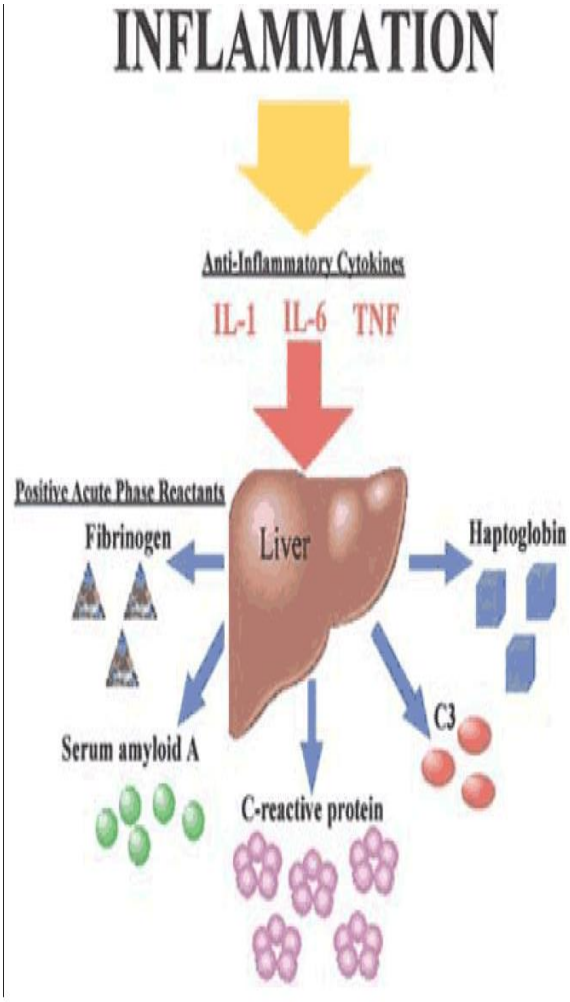


Figure 6: synthesis of CRP[47]

CRP has been recognized as one of the most sensitive **, 'Acute phase proteins' the** concentrations of which in plasma usually rises intensely after

(a) Myocardial infarction

(b) Trauma

(c) Stress

(d) **Infection**

(e**) Inflammation**

(f) Neoplastic proliferation or surgery.[53]

The increase in concentration of CRP begins after 6 to 12 hours of the onset of any of these disorders, and may reach 2000 times than the normal value.[51]

Measurement of CRP is clinically useful for

(A) Screening for many organic diseases

(B) Assessment of the activity about inflammatory diseases

(C) Finding of inter current infections in Systemic Lupus Erythematosis

(D) (SLE), Leukemia, or After surgery (secondary rise in plasma concentration)

(E) **Management of neonatal septicemia and meningitis**[54] in which specimen collections for bacteriological investigations may be difficult.

LABORATORY METHODS[51]

There are three types of method used for measurement of CRP

1.Qualitative method

2.Semiquantitative method

3.Quantitative method

QUALITATIVE METHOD

Latex agglutination qualitative method is a first method used for determination of CRP in patient sera.[55,56,57]

PRINCIPLE

Latex reagent which contain polystyrene coated with anti - human CRP antibodies, reacts with CRP which is present in test sample forming CRP antibody complex which can form precipitates.

PROCEDURE

Test serum is taken in a glass test tube or slide in which latex reagent is added and be observed for any signs of agglutination

INTERPRETATION

- This test is used to determine presence or absence of CRP in given sample by agglutination reaction[55,56]
- CRP Positive: Presence of agglutination and indicates ,concentration of CRP >6mg/L or >10 mg/L
- CRP Negative: Absence of agglutination which indicates a concentration of CRP <6mg/L or <10 mg/L[55,59]

ADVANTAGE

Used as bed side test and result will be available witthin 10 – 15 minutes[59]

LIMITATIONS

- Low sensitivity,
- Positive test will occur in any inflammatory condition
- Positive qualitative test should be followed by semiquantitaive test which is more sensitive than qualitative method.[55,56,59]

SEMI - QUANTITATIVE METHOD

Principle: this method share the same principle which is mentioned in qualitative method

PROCEDURE

Serial dilution of sample is prepared and latex reagent is added to each diluted sample and examined for presence of agglutination.[56,59,60]

INTERPRETATION

Agglutination seen in highest diluted serum corresponds to an approximate amount CRP which is reported as an ratio or concentration of CRP in mg/dL or mg/L as follow[58,60]

1:6 ratio = 6 -12 mg/L

1:12 ratio = 12to 24 mg/L

1:24 ratio = 24to 48 mg/L,

1:48 ratio >48mg/L

QUANTITATIVE METHOD

Quantitative measurement of CRP is the most rapid and sensitive method and concentration of CRP usually reported in mg/L or mg/dL[55,61,62,63]

PRINCIPLE

Diluted serum samples are added with a reagent which contain monoclonal antibodies that react to CRP which is present in test sample. Finally CRP- antibody complexes are measured.

CRP is normally present in plasma at low levels, and so sensitive Immunochemical methods are required for its quantitation which includes

(a) Elisa method

(b) Immunofluorescence method

(c) particle-enhanced Immunoturbidimetry or Nephelometry,

(d) Immunochemiluminescence

and agar or cellulose acetate gel electrophoresis can used for its quantitation.

a)ELISA METHOD

In this method test sample which contain CRP reacts with enzyme marked anti -CRP antibodies producing enzyme marked anti -body-CRP complex which is measured by spectrophoto meter[61]

b)**IMMUNUO FLOURESCENCE METHOD**

In this method test sample which contain CRP reacts with fluorescent marked anti CRP antibodies producing fluorescent marked anti body-CRP complex which is measured by fluorescent microscope

c)IMMUNOTURBIDIMETRY AND NEPHELOMETRY

Both these methods directly measure the precipitation or agglutination caused by antibody –CRP complex

Immunoturbidimetry:

In this method a beam of light is passed through the test cuvette then amount of light transmitted is measured which is directly proportional to the concentration of CRP

Immunonephelometry:

In this method a beam of light passed through the test cuvette ,then amount of scattered light is measured which corresponds to concentration of CRP present in the sample.[53,62,63,64]

CRP and NEONATAL SEPSIS

Any elevation of CRP in the serum of newborn denotes endogenous synthesis because it crosses the placenta in very little amounts[65,66]. In blood CRP rises after 6 to 12 hours, following onset of infection and some time it may take even up to 24 hours to rise after attack of infection. So CRP concentration should not be checked immediatly at birth or at initial manifestation of symptoms suggestive of sepsis.

In early onset sepsis (EOS-before 72 hours old), measurement of single CRP with in 24 hours of illness has a 93% sensitivity for "probable" sepsis.So 2nd measurement of CRP after 24 hours are better for diagnosis[67].

In LOS the dependability of the test is similarly high. A single CRP level at 24-48 hours following the onset of infection has a sensitivity of 85%.[67]

The positive predictive(PPV) value for a high CRP value is poor predictor of sepsis. However, the negative predictive value(NPV) is 93% and therefore it helps to make decision to stop treatment.[68]

CRP level about10 mg/L has been regularly shown to be the most reliable cut-off level to indicate sepsis. There is a association between increased concentration of CRP and the risk of sepsis, with positive predictive value(PPV) gradually increasing up to CRP >100 mg/L.[69]

A lower CRP level was reported in preterm babies compared to term newborns following infection resulting in lower sensitivity.[69]

Many studies reported the magnitude of the CRP response to sepsis depends on underlying pathogen. Low rise of CRP is reported in coagulase negative staphylococci compared to Staphylococcus aureus, group B streptococci, and E.coli.[69]

Serial CRP measurement is helpful to monitor treatment of neonates whose CRP levels fail to decrease, or continue to increase after 48 hours of antibiotic therapy suggest treatment failure.[65,70,71]

NON INFECTIOUS CONDITIONS IN NEONATES AND CRP ELEVATION

Many studies reported CRP elevation associated with certain non infectious conditions. [72, 73] They are listed below

1 Prolonged rupture of membranes[74, 75]

2. Maternal fever during labour [74 ,75]

3. Fetal distress [74]

4. Perinatal asphyxia[74,75,76]

5. Shock,

6. Intraventricular haemorrhage[76]

7. Pneumothorax[76]

8. Meconium aspiration pneumonitis.[74,75,76]

CELLULAR PARAMETERS

CBC can be used in determining the changes associated with infection .It is also used for monitoring the treatment of any infection. Such monitoring of the CBC in neonatal sepsis may be useful in assisting the differentiation of sepsis from nonspecific abnormalities due to the stress of delivery.

Among CBC ,Total WBC count (which means total number of white blood cell in a micro litre of blood) and differential count (percentage of each type of white blood cell) are commonly used as cellular parameters for analysing sepsis.

WHITE BLOOD CELLS (WBC)

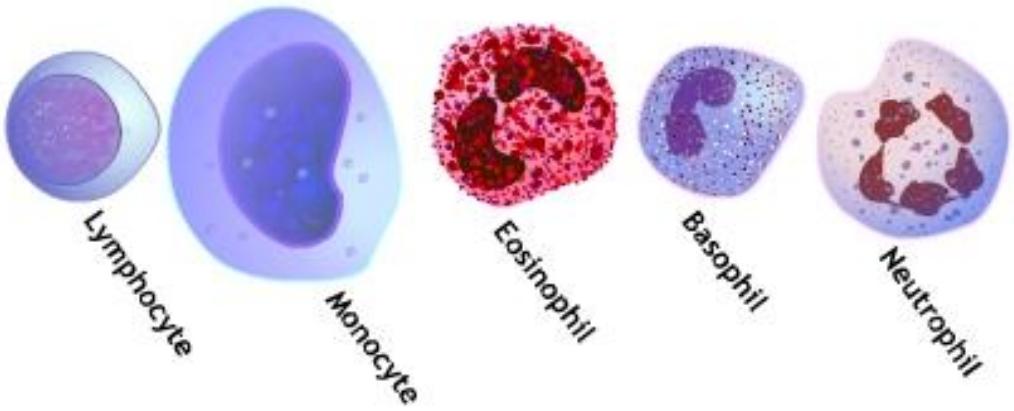


Figure 7 -Types of WBC

White blood cells (Leukocytes) can be classified into two types- granulocytes and agranulocytes.

Granulocytes: Leukocytes which contain granules in their cytoplasm is called granulocytes.

Examples:

Neutrophils

Eosinophils

Basophils

They are also called polymorphonuclear leukocytes (PMN) because they have multilobular nucleus. Among them neutrophil have segmented nucleus , so it is called segmentedneutrophil.

Agranulocytes:

Leukocytes which do not contain granules in their cytoplasm is called agranulocytes.

Examples

Lymphocytes and monocytes

They have non lobular nucleus, so they are called mononuclear leukocytes.

FUNCTIONS OF WBC

Normal lifespan of white blood cells ranges about 13 to 20 days, after that they are destroyed in the lymphatic system. When immature WBCs are released from the bone marrow into the peripheral blood, they are called as band cell.

Leukocytes fight against infection by phagocytosis. During phagocytosis, leukocytes engulf invading pathogen and other foregin material and destroy them. They also produce, carry, and distribute antibodies as part of the body's immune response.

WBC COUNT, ABSOLUTE NEUTROPHIL COUNT, AND NEONATAL SEPSIS

Newborn has a high white blood cell count at birth, ranging from 9,000 to 30,000 leukocytes. This number gradually falls to adult levels within two weeks. Neutrophil percentage of newborn in first week of life is very high but after that lymphocytes are predominant cells up to 8 years of age[77].

Total leukocyte count (TLC)(percentage of lymphocyte + monocyte/PMNs + bands) is less sensitive for determining sepsis compared to absolute neutrophil count (mature neutrophil and immature cells) which is slightlymore sensitive than TLC.

Only two thirds of neonates have abnormal neutrophil counts at the time of onset of symptoms. Therefore the neutrophil count may not provide satisfactory confirmation of sepsis. Neutropenia is also noted in maternal hypertension, periventricular or intraventricular hemorrhage and severe perinatal asphyxia.

I/T RATIO AND NEONATAL SEPSIS

I/T ratios can be used in diagnosing neonatal sepsis. Sensitivity of Immature-to-total (I/T) ratio is used to determine the sepsis and is about(60-90%)[78]. In neonates I/T ratio >0.2 is used to determine sepsis .I/T ratio of 0.16 is maximum acceptable ratio which excludes sepsis s in the first 24

hours. In utmost newborns, the ratio decreases to 0.12 within 60 hours of birth[78].

Increased I/T ratios may be noted in other physiological events which limit the positive predictive value of I/T to diagnose the sepsis. so elevated I/T ratio could be used in combination with other markers and clinical signs[78].

PLATELET COUNT AND NEONATAL SEPSIS

Normal platelet count of neonates is usually ≥150,000 /µL In healthy newborn. This value rarely goes lower than 100,000/µL in the first 10 days of life.Thrombocytopenia (decreased plate count < 100,000/µL) has been one of the sign of sepsis and it present as long as 7 days and 10-60% of Infants present with sepsis have thrombocytopenia[78]

AIM

This study is aimed to evaluate CRP and cellular parameters (WBC count,Absolute Neutrophil Count(A.N.C), Immature and total neutrophil ratio (I/T Ratio,platelet counts) along with clinical parameters to diagnose the neonatal sepsis.

PRIMARY OBJECTIVE

1. The purpose of this study is to verify the utility of CRP and cellular markers in early diagnosis of neonatal sepsis by their sensitivity specificity, Positive Predictive value(PPV),and negative predictive value(NPV) in comparison with blood culture.
2. To compare CRP with cellular parameters (WBC,ANC,I/T ratio and platelet count) in neonates with risk of sepsis
3. To compare CRP with clinical parameters.

MATERIALS AND METHODS

The cross sectional study was conducted in NICU, Department of paediatrics, Department of biochemistry, RSRM ,Government Stanley medical college and Hospital over the period of 6months (FEB 2017 –JULY 2017). Ethical approval was taken from ethical committee before initiation of the study.The study was conducted in148 neonates admitted in NICU with risk of sepsis (study subject) which includes clinical signs and symptoms suggestive of sepsis or neonates presenting with maternal risk factors of sepsis. Based on following inclusion and exclusion criteria,the neonates were enrolled in the study after informed consent was obtained from parents/ guardian.

INCLUSION CRITERIA

According to WHO integrated criteria for risk of sepsis ,neonates who were admitted in NICU with following 2 or more criteria were recruited in this study[78]

1. Temperature instability (Temperature > 37.5° or <35.5°)
2. Tachycardia (heart rate measurement >180 /min by auscultation/min)
3. Bradycardia (heart rate < 90 /min by auscultation method)
4. Respiratory distress (Respiratory rate >60 /min by visual & abdominal palpation)
5. Poor feeding (Not doing sucking properly, number of feeds less than 8 times /day)

6. Irritable (cry incessantly)
7. Lethargy (Lethargic babies have little or no energy. They sleep longer than normal, and it is difficult to wake them for feedings. They are drowsy or sluggish; they are not alert and they do not pay attention to visual stimulation or sounds).
8. Abdominal Distension
9. Poor Capillary Refilling Time (CRT) >3sec
10. Maternal fever
11. Premature rupture of membrane(PROM)
12. prolonged premature rupture of membrane(>18 hours)
13. MSAF(meconium stained amniotic fluid)
14. Birth asphyxia
15. Low Apgar score score(<7)

EXCLUSION CRITERIA

1. Neonates born with congenital anomalies
2. Neonates receiving antibiotics before investigation
3. Neonates already received treatment outside and referred to our institution.

Detailed history and clinical findings of each neonate was recorded in the study proforma(Neonatal Case Record Sheet).In accordance with the NICU's routine procedure, under strict aseptic precaution

blood samples were collected for the following laboratory parameters. Then following tests were done.

1. CRP
2. White Blood Cell Count (WBC)
3. Absolute Neutrophil Count (ANC)
4. Immature/Total neutrophil ratio (I/T Ratio)
5. Platelet count
6. Blood culture

LABORATORY INVESTIGATION :

ESTIMATION OF CRP

According to the time of sample collection CRP was classified into CRP1 and CRP2.

CRP1: Time of sample collection after 6 hours of clinical suspicion of sepsis

CRP2: Time of sample collection after 48 hours of clinical suspicion of sepsis

2 ml of blood was collected in red top tube for CRP estimation. It was allowed to clot for 20 minutes ,then serum was separated and centrifuged at 2000 –2500 rpm for 15 minutes.

Then serum was separated and estimated using Semi auto analyser by Immuno turbidimetric method.

METHOD: Immuno turbidimetric method

PRINCIPLE:

Test sample containing CRP reacts with reagents which contain latex particles coated with antibodies to human CRP causing agglutination which is measured against 540 nm

REAGENTS COMPOSITION:

Reagent 1 : Diluent- Tris buffer solution (pH 8.2)

Reagent 2: Suspension of Latex particles coated with goat IgG anti –human CRP

Concentration of reagent 0.2% w/n

Working Reagent: 1 mL Latex Reagent + 9 mL Diluent

SYSTEM PARAMETER

Mode	Fixed Time
Reaction	Ascending
Wavelength	540 nm
Blank with	Distilled water
Sample volume	5 µL
Reagent volume	1000µL
Delay time	5 sec
Read time	120 sec
No of reading	2
Linearity limit	150 mg/L
Unit	mg/L

LABORATORY PROCEDURE:

	Standard	Sample
Standard	5 µL	-
Sample	-	5 µL
Reagent	1000 µL	1000 µL

1.preparation of calibrator

- Working reagent 1ml
- calibrator 5µL
- calibrator was mixed with reagent and the absorbance was read immediately(A1 calibrator)

2.Preparation of sample for test

- Working reagent 1ml
- sample 5 µL
- Calibrator was mixed with reagent and the absorbance was read immediately(A1 sample)

3.Incubated at 37°c

After 2 minutes read sample absorbance (A2sample) and calibrator absorbance(A2 calibrator) was read

CALCULATION:

(A2-A1) sample / (A2- A1) calibrator x Calibrator concentration = CRP mg/L of sample

BLOOD CULTURE:

Under aseptic precaution 1 ml blood was collected in culture bottle at the time of clinical suspicion of sepsis. Blood culture bottle contains Brain Heart Infusion broth (BHI) in a ratio of blood to BHI of 1:10. Subsequent sub-culture was done on day 1, 3 and 7 on 5% sheep blood agar, chocolate agar and MacConkey agar . Identification of bacteria was performed according to the Clinical Laboratory standard Institute (CLSI) guidelines. Blood cultures were defined as positive when the same microorganism with the same antimicrobial sensitivity grew in both samples within 72 hours of collection.

CELLUAR PARAMETER (WBC COUNT,ABSOLUTE NEUTROPHIL COUNT,I/T RATIO, PLATELET COUNT)

1ml of blood was taken in EDTA tube at time of clinical suspicion of sepsis and estimated by sixpart Automated Sysmex haematology analyser.

Principle:

Basic principle of sysmex haematology analyser based on flow cytometry method using a semiconductor laser. A two dimensional scatter gram is plotted.

x –axis of scatter gram representing the intensity of side fluorescent light and y-axis representing the intensity of forward scattered light. and displays white blood cells, platelets, red blood cells, Immature cells, nucleated RBC cells.

There are various channels used for detecting WBC, Immature cells, RBC, platelets.

WDF channel

Used for White Blood Cell such as neutrophils, lymphocytes, monocytes,eosinophil basophils and debris.

WPC channel

Used for detecting Immature cells.

PLT channel

Used for detecting platelets

RBC channel

Used for detecting red blood cells

CUT OF VALUE FOR EACH PARAMETER

1. CRP : >5 mg/L. (Normal range 2-5 mg/L)
2. WBC Count : <5000/µL or >15000/µL (Normal range 5000-15000µL)
3. ANC :<1800/µL (Normal range 1800- 8000 µL)
4. I/T ratio : >0.2 (Normal value < 0.1)
5. Platelet count :<150000/µL (Normal range1.5-4Lakes/ µL).

STATISTICS AND RESULTS

In this study, 148 neonates with risk factors of sepsis were evaluated. CRP(CRP1&2), cellular parameters (WBC count, ANC,I/T ratio, Platelet count),and blood culture were performed in all neonates with risk of sepsis. All clinical data and results of CRP ,cellular parameters and blood culture were entered in excel sheets and statistical analysis done by using SPSS software.

CUT OF VALUE FOR EACH PARAMETER

1. CRP :>5 mg/L. (Normal range 2-5 mg/L)
2. WBC Count : <5000/µL or >15000/µL (Normal range 5000-15000µL)
3. ANC :<1800/µL (Normal range 1800- 8000 µL)
4. I/T ratio :>0.2 (Normal value < 0.1)
5. Platelet count :<150000/µL (Normal range1.5-4Lakes/ µL).

Based on blood culture results , neonates were divided into two groups

1. Culture positive sepsis (proven sepsis) - culture positive results plus 2 or more risk factors of sepsis.
2. Culture negative sepsis (probable sepsis) –Culture negative results plus 2 or more risk factors of sepsis.

Sensitivity, specificity,PPV,NPV of each parameter was compared with gold standard test(blood culture)

Biochemical parameters CRP 1, CRP 2 and cellular parameters were compared with culture proven and probable sepsis along with clinical parameters.

1. Chi-square test was used to compare CRP and cellular parameters with culture proven and probable sepsis
2. Chi-square test was used for comparing the cellular parameters between CRP1 and CRP2 positive cases and negative cases
3. Diagnostic odds ratio,z test used for comparing the relative risk of clinical parameters with proven and probable sepsis and comparing the clinical parameters with CRP1,CRP2 positive and negative cases. A p-value of <0.05 considered as statistically significant

TABLE 1: BASELINE CHARACTERISTICS OF THE STUDY POPULATION:

	No of Cases	**Percentage**
Gender		
Male babies	90	60.81 %
Female babies	58	39.29 %
Mode Of Delivery		
Normal	63	42.56 %
Assisted	85	57.44 %
Maturity		
Term	67	45.27 %
Pre-term	81	54.72 %
Birth Weight		
Low	76	51.35 %
Normal	72	48.64 %
Culture		
Positive	53	35.81 %
Negative	95	64.18 %

Table 1: Displays the baseline characteristics of the study population .It shows Out of 148 neonates

1. 90(60.8%) were male and 58 (39.2%) were female.

2. 85 (57.44%)were delivered through assisted deliveries ,and 63(42.5%) were delivered through vaginal route.

3.Based on maturity 81(54.7%) were pre term babies ,67 (45.2%) were term babies.

4.Regarding birth weight 76(51.3%) were Low birth weight(LBW),72 (48.6%) were normal weight.53(35.8%) were

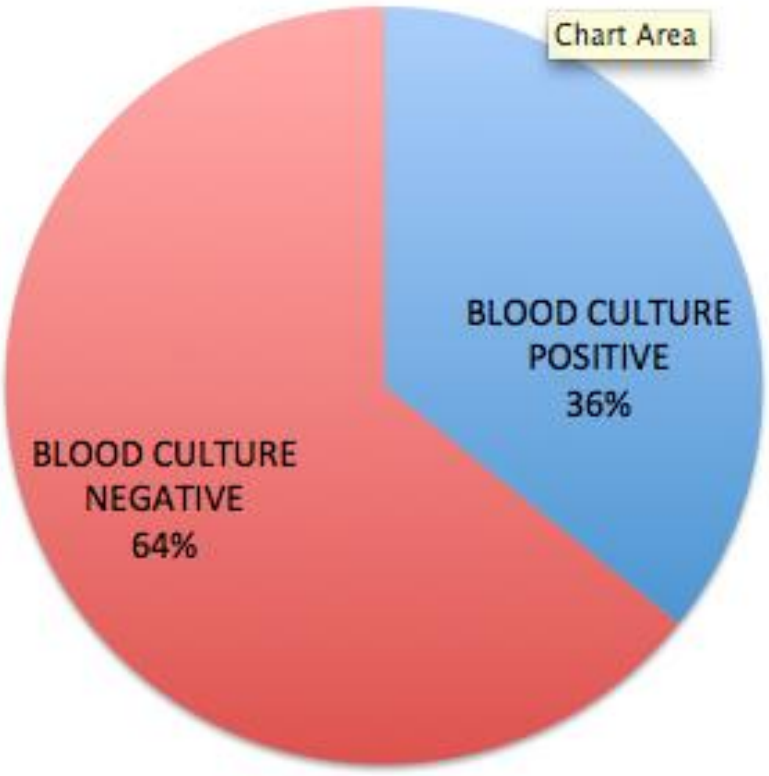
5. blood culture positive,95 (64.1%) were negative on blood culture.

Table -2 COMPARISON OF BASELINE CHARACTERS OF NEONATES IN CULTURE POSITIVE AND CULTURE NEGATIVE SEPSIS

	Culture positive Sepsis n=53		**Culture negative sepsis n=95**	
	Number	%	Number	%
Gender				
Male	45	84 %	50	52.6 %
Female	8	16 %	45	47.4 %
Maturity				
Pre-term	35	66 %	46	48.4 %
Term	18	34 %	49	51.6 %
Mode of Delivery				
Assisted	21	38.1 %	64	67.3 %
Normal	32	61.9 %	31	32.7 %
Birth Weight				
Low	36	67 %	40	42.1 %
Normal	17	23 %	55	57.9 %

Table 2 explains percentage of male neonates preterm babies LBW babies were high in culture proven sepsis compared with culture negative sepsis which was statistically significant p vale <0.05

GRAPH -1 PRECENTAGE OF CULTURE POSITIVE AND NEGATIVE NEONATES



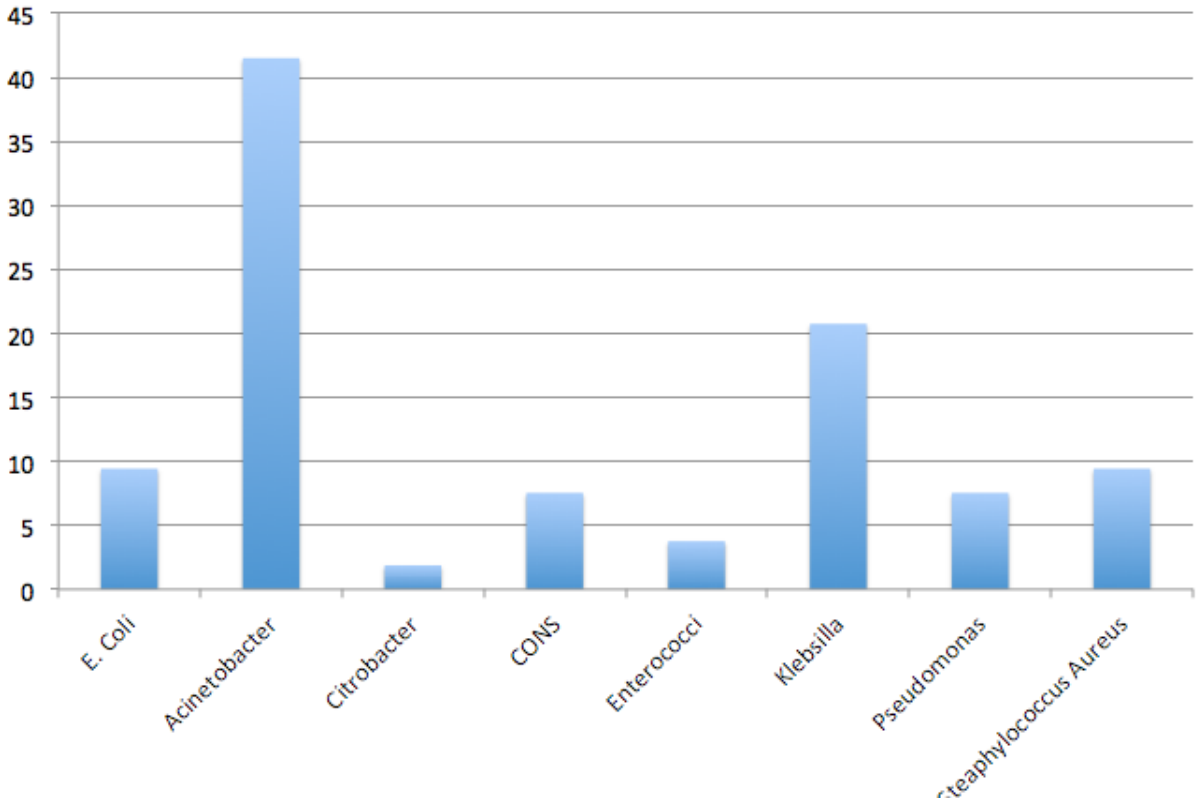
GRAPH 1 shows percentage of culture positive cases are less compared with culture negative neonates.

TABLE 3-MICRO ORGANISM FOUND IN BLOOD CULTURE REPORTS

MICRO ORGANISM FOUND IN BLOOD CULTURE	NO OF PATEINTS	%
ECOLI	5	9.43%
ACINETOBACTER	22	41.50%
CITRO BACTER	1	1.88%
COAGULASE NEGATIVE STAPHYLOCOCCUS	4	7.54%
ENTEROCOCCI	2	3.77%
KLEBSIELLA PNEUMONIAE	11	20.75%
PSEUDOMONAS AERUGINOSA	4	7.54%
STAPHYLOCOCCUS AUREUS	5	9.43%
TOTAL	53	100%

- Table 3 and graph 2 shows percentage of microorganism found in blood culture
- Acinetobacter was the most common organism isolated followed by Klebsiella and Coagulus negative Staphylococcus aureus , Staphlo coccus aureus
- Enterobacter, Pseudomonas , E.coli citrobacter were less commonly present

Graph 2 –profile of microorganism isolated from blood sample (n=53)



X axis- shows various microorganism organism found in blood culture.

Y axis –shows percentage of microorganism found in blood culture.

Table 4 Clinical Parameters of Study Subject

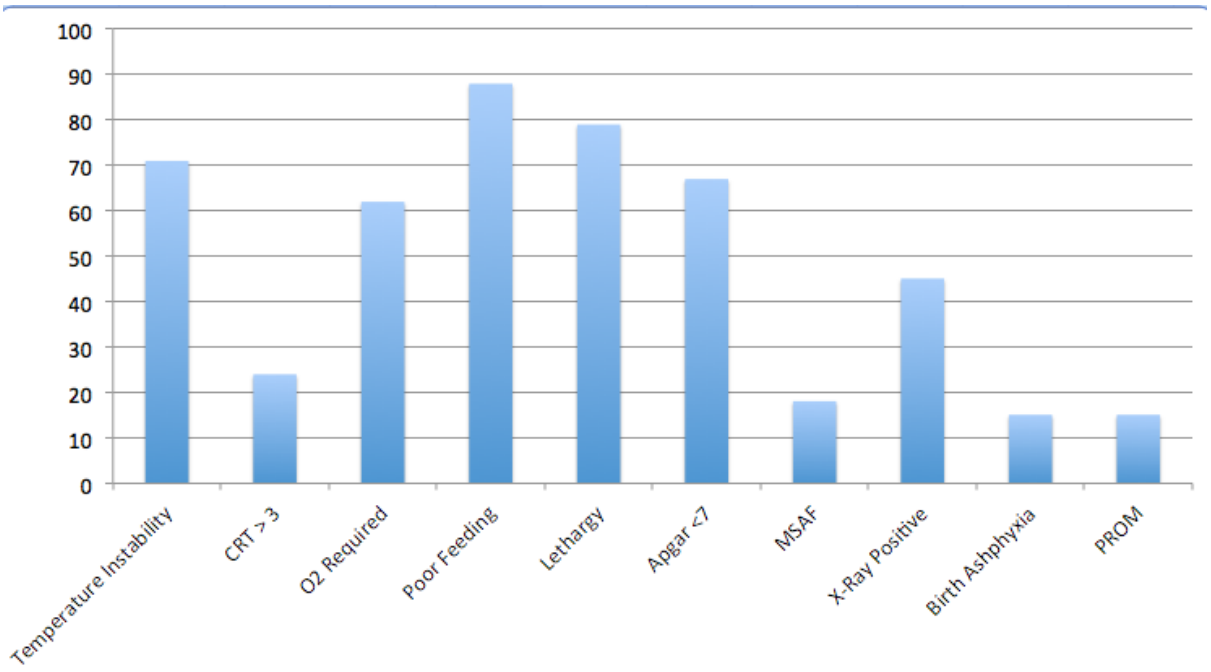
	No of Cases	**Percentage**
Temperature Instability		
< 34.5 or > 37.5	58	39.18 %
34.5 to 37.5	90	60.81 %
CRT		
>3	24	16.21 %
<3	124	83.78 %
O2 Required		
Yes	58	39.18 %
No	90	60.81 %
Feeding		
Poor	92	62.16 %
Normal	56	37.83 %
Lethargy		
Yes	85	57.43 %
No	63	42.56 %
Apgar Score		
<7	38	25.67 %
>7	110	74.32 %
X-ray Finding		
Positive	35	23.64 %
Negative	113	76.36 %
MSAF		
Present	17	11.48 %
Absent	131	88.52 %
Birth Asphyxia		
Present	16	10.81 %
Absent	132	89.19 %
PROM		
Present	11	7.43 %
Absent	137	92.57 %

Table 4 explains number and percentage of Clinical parameters of all neonates enrolled in this study .

It shows Out of 148 cases temperature instability was present in 58 (39.1%) neonates ,Capillary refilling time was increased (CRT>3) in 24 neonates (16.2%), neonates requiring oxygen for their respiratory distress were 58(39.1%),92 (62.1%) had poor feeding,85 (57.4) were lethargic ,38 (25.67 %) had low Apgar score (<7) , MSAF (Meconium stained amniotic fluid was present in 17 (11.4%)neonates.35(23.6%) had positive x ray finding suggestive of sepsis .16 (10.8%) neonates had history of birth asphyxia, 11 (7.4%) were presented with history of premature rupture of membrane (PROM.)

Table 5- explains % of each clinical parameters presented in culture positive sepsis

Positive Clinical Parameters	Culture Proven Sepsis n=53	
	Number	**%**
Temperature Instability	38	71 %
CRT > 3	13	24 %
O2 Requiring neonates	33	62 %
Poor Feeding	47	88 %
Lethargy	42	79.3 %
Apgar Score	36	67 %
MSAF	10	18.1 %
Positive X-ray Finding	24	45.1 %
Birth Asphyxia	8	15.1 %
PROM	8	15.1 %



Graph-3 explains % of clinical parameters presented in culture positive sepsis n=53

Table 6 - Comparison of CRP1 with Blood Culture

CRP1	Culture +ve	Culture -ve	Total	Chi -square value	P value
Positive (>5 mg/L)	44	53	97	9.99	**<0.001** ***Statistically Significant***
Negative (<=5 mg/L)	9	42	51		
Total	53	95	148		

- Table 6 shows CRP 1 was positive (>5mg/L) in 97 (65.54%) neonates out of 148 cases.
- CRP 1 was negative (<5mg/L) in 51 (34.45%) neonates out of 148 case.
- Out of 53 positive blood culture CRP 1 was positive in 44 (83%) neonates
- Out of 95 negative blood culture crp1 was positive in 53 (55.7%) neonates
- So association between CRP1 and Blood culture results were statistically
- Significant with p value <0.0001

Table 7 Comparison of CRP2 with Blood Culture

CRP2	**Culture +ve**	**Culture -ve**	**Total**	**Chi-square value**	**P value**
Positive (>5 mg/L)	51	57	108	20.83	**<0.001**
Negative (<=5 mg/L)	2	38	40		***Statistically Significant***
Total	53	95	148		

- Table 7 shows CRP 2 was positive (>5mg/L) in 108 (72.9%) neonates out of 148 cases.
- CRP 2 was negative (<5mg/L) in 40 (27.0%) neonates out of 148 case.
- Out of 53 positive blood culture CRP 2 was positive in 51 (96.2%) neonates
- Out of 95 negative blood culture crp2 was positive in 57 (60%) neonates
- Association of CRP2 with blood culture results were statistically significant with p value <0.0001

Comparison of WBC with Blood Culture

Table 8

WBC	Culture +ve	Culture -ve	Total	Chi -square value	P value
Positive (<5000 or >15000 µL)	33	45	78	2.46	**0.1** ***Statistically Not Significant***
Negative (5000 – 15000 µL)	20	50	70		
Total	53	95	148		

- Table 8 shows WBC was positive (<5000/µL or >15000 µL) in 78 (52.7%) neonates out of 148 cases.
- WBC was negative (5000-15000 µL) in 70 (42.3%) neonates out of 148 case.
- Among of 53 positive blood culture WBC was positive in 33 (62.3%) neonates
- Out of 95 negative blood culture WBC was positive in 57 (47.3%) neonates
- This test result was not statistically significant with p value =0.1

Comparison of ANC with Blood Culture

Table 9

ANC	Culture +ve	Culture -ve	Total	Chi -square value	P value
Positive (<1800 µL)	1	3	4	0.20	**0.6** ***Statistically Not Significant***
Negative (>1800 µL)	52	92	144		
Total	53	95	148		

- Table 9 shows ANC was positive (<1800 /µL) in 4 (2.7%) neonates out of 148 cases.
- ANC was negative (>1800µL) in 144 (97.2%) neonates out of 148 cases.
- Among the 53 positive blood culture cases ANC was positive in 1 (1.9%) neonate
- Out of 95 negative blood culture cases ANC was positive in 3 (3.1%) neonates
- P value of this test result was 0.64 (not statistically significant)

Comparison of I/T with Blood Culture

Table 10

I/T	Culture +ve	Culture -ve	Total	Chi -square value	P value
Positive (>0.2)	10	1	11	13.2	**0.0003** ***Statistically Significant***
Negative (<0.2)	43	94	137		
Total	53	95	148		

- Table 10 shows I/T was positive (.>0.2) in 11 (7.4%) neonates out of 148 cases.
- I/T was negative (<0.2) in 137(92.6%) neonates out of 148 cases.
- Among the 53 positive blood culture cases I/T was positive in 10 (18.9%) neonates
- Out of 95 negative blood culture cases I/T was positive in 1 (1.05%) neonate.
- This test result showed statistically significant p value =0.0003

Comparison of Platelet with Blood Culture

Table 11

Platelet	**Culture +ve**	**Culture -ve**	**Total**	**Chi -square value**	**P value**
Positive (<1.5 lakhs)	8	16	24	0.002	**0.9** ***Statistically Not Significant***
Negative (>1.5 lakhs)	45	79	124		
Total	53	95	148		

- Platelet count (PLT) was positive (<I.5 Lakes) in24 (16.2%) neonates out of 148 cases.
- PLT was negative (>1.5 Lakes) in 124 (83.7%) neonates out of 148 cases.
- Among of 53 positive blood culture PLT was positive in 8 (15%) neonates
- Out of 95 negative blood culture PLT was positive in 16 (20.2%) neonates
- P value was 0.9 and statistically not significant

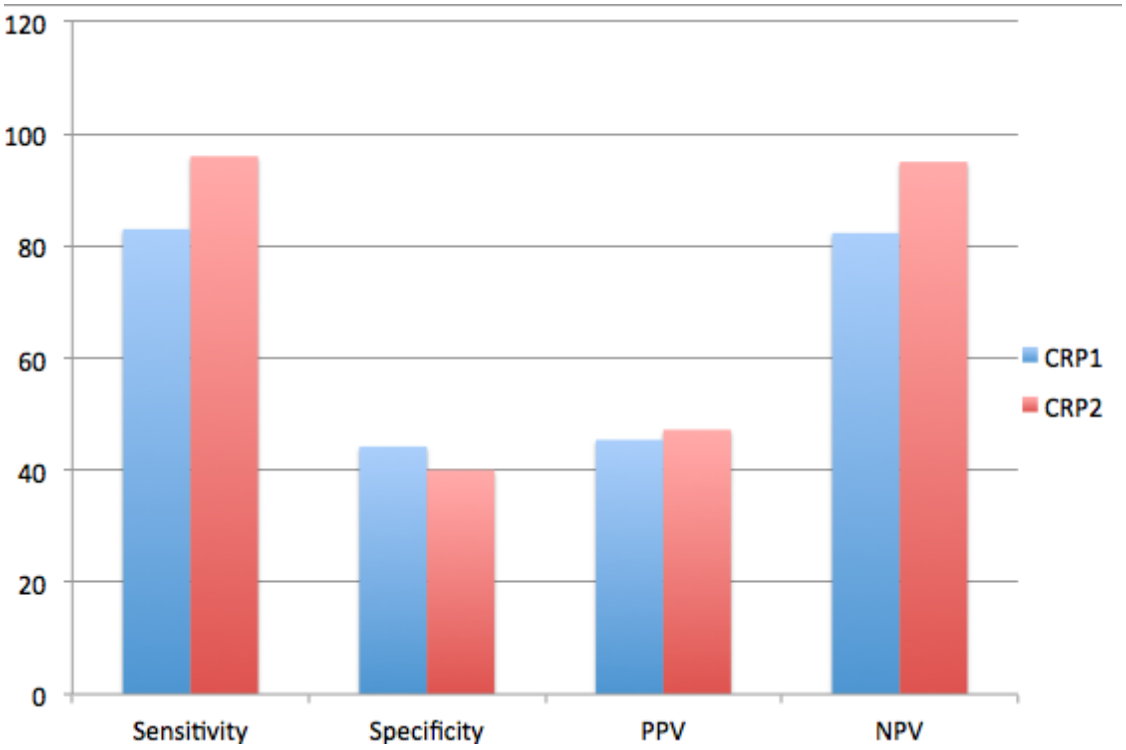
Sensitivity, Specificity, PPV and NPV of CRP and cellular Parameters

Table 12

	Sensitivity%	**Specificity%**	**PPV%**	**NPV%**
CRP1	83.0	44.2	45.4	82.3
CRP2	96	40	47,2	95
WBC	62.2	52.6	42.3	71.4
ANC	1.92	92	25	63
I/T	11.3	94.7	54.5	65.7
Platelet	17	83.1	33.3	63.7

- Table 12 and graph 4 shows CRP2 has highest sensitivity 96% and NPV 95%, next to this CRP1 has 83% sensitivity and NPV 82.3%
- I/T has highest specificity 94.73% , PPV 54.5% next to this ANC has 92%Specificity ,CRP1 has 47.2% PPV

GRAPH 4



Graph 6 shows sensitivity ,specificity ,PPV,NPV of CRP 1&2 in x axis

Y axis shows the percentage.

Table 13,14,15, 16- Comparison of CRP1 with Cellular Parameters

TABLE 13 :CRP1 Vs WBC

WBC	CRP1 +ve	CRP1 - ve	Total	Chisquare value	P value
Positive (<5000 or >15000 µL)	65	13	78	21.4	**<0.0001** ***Statistically Significant***
Negative (5000 – 15000 µL)	32	38	70		
Total	97	51	148		

Table 13 shows comparison of CRP1& WBC

CHI-SQUARE VALUE-21.4

P <0.0001 (Statistically Significant)

CRP1 Vs ANC

Table 14

ANC	CRP1 +ve	CRP1 - ve	Total	Chisquare value	P value
Positive (<1800 µL)	3	1	4	0.16	**0.6** ***Statistically Not Significant***
Negative (>18000 µL)	94	50	144		
Total	97	51	148		

Table 14 shows comparison of CRP1& ANC

CHI-SQUARE VALUE-0.163

P =0.68 (NOT Significant)

CRP1 Vs I/T RATIO

Table 15

I/T	CRP1 +ve	CRP1 - ve	Total	Chi -square value	P value
Positive (>0.2)	11	0	11	4.7	**0.03** ***Statistically Significant***
Negative (<0.2)	86	51	137		
Total	97	51	148		

Table 15 shows comparison of CRP1& I/T RATIO CHI-SQUARE VALUE-

4.708

P =0.03 (Significant)

CRP1 Vs Platelet

Table 16

Platelet	CRP1 +ve	CRP1 - ve	Total	Chisquare value	P value
Positive (<1.5 lakhs)	15	9	24	0.01	**0.9** ***Statistically Not Significant***
Negative (>1.5 lakhs)	82	42	124		
Total	97	51	148		

Table 16 – shows comparisonCRP1&PLATELET COUNT

CHI-SQUARE VALUE-0.012

P =0.91 (NOT Significant)

CRP2 Vs WBC

Table 17

WBC	CRP2 +ve	CRP2 -ve	Total	Chisquare value	P value
Positive (<5000 or >15000 µL)	68	10	78	15.39	**<0.0001** ***Statistically Significant***
Negative (5000 – 15000 µL)	40	30	70		
Total	108	40	148		

Table 17 - shows comparision of CRP 2& WBC

CHI-SQUARE VALUE-15.4

P = <0.0001 (Significant)

CRP2 Vs ANC

Table 18

ANC	CRP2 +ve	CRP2 -ve	Total	Chisquare value	P value
Positive (<1800 µL)	3	1	4	0.006	**0.9** ***Statistically Not Significant***
Negative (>18000 µL)	105	39	144		
Total	108	40	148		

Table 18 – shows comparisonCRP2&ANC COUNT

CHI-SQUARE VALUE-0.0006

P =0.9 (NOT Significant)

CRP2 Vs I/T

Table 19

I/T RATIO	CRP2 +ve	CRP2 - ve	Total	Chisquare value	P value
Positive (>0.2)	11	0	11	3.045	**0.08** ***Statistically Not Significant***
Negative (<0.2)	97	40	137		
Total	108	40	148		

Table 19- shows comparisonCRP2&I/T

CHI-SQARE VALUE-3.04

P =0.08 (NOT Significant)

CRP2 Vs Platelet

Table 20

Platelet	CRP2 +ve	CRP2 - ve	Total	Chisquare value	P value
Positive (<1.5 lakhs)	22	2	24	4.0	**0.04** ***Statistically Significant***
Negative (>1.5 lakhs)	86	38	124		
Total	108	40	148		

Table 19- shows comparison CRP2&PLATELET COUNT

CHI-SQUARE VALUE-0.012

P =0.045 (Significant)

Table 21: Comparison of Clinical Parameters with Culture Result

CLINICALPARAMETRS	ODDS Ratio	95 % CI	Z value	P value
Temperature Instability	9.737	4.45 -21.28	5.7	<0.0001 *Statistically Significant*
CRT	2.48	1.02- 6.02	2.0	0.04 *Statistically Significant*
O2 Required	4.62	2.5 – 9.48	4.17	<0.0001 *Statistically Significant*
Poor Feeding	8.70	3.39 – 22.2	4.5	<0.0001 *Statistically Significant*
Lethargy	5.5	2.55 -11.8	4.3	<0.0001 *Statistically Significant*
Apgar Score	9.27	3.96 – 21.6	5.13	<0.0001 *Statistically Significant*
Mode of Delivery	0.31	0.15 – 0.63	3.21	0.0013 *Statistically Significant*
Maturity	2.07	1.03 – 4.15	2.04	0.04 *Statistically Significant*
Birth Weight	2.38	1.16 – 4.8	2.37	0.01 *Statistically Significant*
PROM	5.45	1.37 – 21.5	2.41	0.01 *Statistically Significant*
Gender	5.06	2.15 – 11.8	3.72	0.0002 *Statistically Significant*
X-ray	2.9	1.04 – 8.2	2.03	0.01 *Statistically Significant*
Birth Asphyxia	1.93	0.68 – 5.4	1.2	0.2 *Statistically Not Significant*
MSAF	2.9	1.04-8.2	2.03	0.04 *Statistically Significant*

TABLE 21 explains COMPARISION OF CLINICAL PARAMETERS WITH BLOOD CULTURE

- Clinical parameters- temperature instability, oxygen requirement, poor feeding, lethargy Apgar <7 are statistically verymuch significant with p value <0.0001)
- Except birth asphyxia all other parameters are statistically significant with blood cultures with p value <0.05

Table 22: Comparison of Clinical Parameters with CRP1

CLINICAL PARAMETERS	ODDS Ratio	95 % CI	Z value	P value
Temperature Instability	2.6	1.2-5.6	2.4	<0.01 *Statistically Significant*
CRT	1.7	0.6-4.6	1.0	0.02 *Statistically Significant*
O2 Required	1.1	0.5-2..2	0.3	0.7 *Statisticall not Significant*
Poor Feeding	2.1	1.0-4.2	2.1	0.03 *Statistically Significant*
Lethargy	72.1	22.9-23	7.3	<0.0001 *Statistically Significant*
Apgar Score	0.8	0.4-1.8	0.3	0.7 *Statistically Not Significant*
Mode of Delivery	2.1	1.0-4.2	2.2	0.0013 *Statistically Significant*
Maturity	2.0	1.02 – 4	2.04	0.04 *Statistically Significant*
Birth Weight	2.1	1.0-4.2	2.2	0.02 *Statistically Significant*
PROM	0.9	0.2-3.2	20.1	0.89 *Statistically not Significant*
Gender	5.06	2.15 – 11.8	3.72	0.29 *Statistically not Significant*
X-ray	5.5	1.8-16.6	3.0	0.002 *Statistically Significant*
Birth Asphyxia	1.1	0.3 – 3.5	0.2	0.7 *Statistically Not Significant*
MSAF	0.9	0.3-2.7	0.07	0.9 *Statistically not Significant*

Table 22: Comparison of Clinical Parameters with CRP1

- Comparison of Clinical parameter with CRP1.Among theseLethargy shows more statistical significance (p value <0.0001)with CRP1
- Other parameters like Temperature instability, Assisted delivery, prematurity ,Low birth weight ,positive X ray finding are also statistically significant with CRP1 (p value <0.05)and CRT, oxygen required Apgar <7 ,PROM, male gender,Birth asphyxia , MSAF are not statistically significant with CRP1

Table 23: Comparison of Clinical Parameters with CRP2

CLINICAL PARAMETERS	ODDS Ratio	95 % CI	Z value	P value
Temperature Instability	4.2	1.7-10.3	3.1	0.001 *Statistically Significant*
CRT	10.5	1.3-80.9	2.2	0.02 *Statistically Significant*
O2 Required	0.7	0.3-1.4	0.8	0.3 *Statistically not Significant*
Poor Feeding	16.6	2.9-14.8	4.6	<0.0001 *Statistically Significant*
Lethargy	15.4	5.8-40.5	5.5	<0.0001 *Statistically Significant*
Apgar Score	7.5	2.1-25.8	3.2	0.0014 *Statistically Significant*
Mode of Delivery	3.0	1.4-6.5	2.9	0.0035 *Statistically Significant*
Maturity	12.8	4.9-33.6	5.2	<0.0001 *Statistically Significant*
Birth Weight	0.3	0.1-0.6	2.8	0.001 *Statistically Significant*
PROM	1.7	0.3 – 8.3	0.6	0.4 *Statistically not Significant*
Gender	0.2	0.07-0.48	3.72	0.0002 *Statistically Significant*
X-ray	17.9	2.3-135	2.03	0.0006 *Statistically Significant*
Birth Asphyxia	2.8	0.6-13.5	1.3	0.1 *Statistically Not Significant*
MSAF	3.06	0.06-14.0	1.4	0.1 *Statistically not Significant*

TABLE 23 SHOWS COMPARISON OF CLINICAL PARAMETERS WITH CRP2

- Prematurity, lethargy, poor feeding, are statistically more significant with CRP2 (p value <0.0001)
- Temperature instability ,CRT>3, Low Apgar score<7 , assisted delivery, LBW, prematurity, positive xray finding are also statistically significant with CRP2 (p value <0.05)
- Oxygen requirement ,PROM ,birth asphyxia, MSAF are not statistically significant with CRP2

DISCUSSION

Neonatal sepsis is a serious life threatening condition and major cause of neonatal mortality and morbidity in developing countries like India. So early diagnosis and treatment is very essential for good out come.Blood culture is the gold standard diagnosis but it is not available in all the peripheral centres, the cost for the test is also high and results are also not available in early period. Blood culture is often negative in many of the neonates in whom clinical signs and symptoms of sepsis is present. Because of these limitations , quick convenient, affordable ,cost effective laboratory method along with clinical parameters is required to evaluate neonatal sepsis. In my study, CRP and cellular parameters (WBC, ANC,I/T Ratio,Platelet count) were used for evaluation of neonatal sepsis along with clinical parameters.

Of 148 neonates in present study 90 neonates (60.8%) were male and 58 (39.2%) were female and 84% of male gender studied in culture proven sepsis compared with culture negative sepsis. So male genders are more affected in neonatal septicemia due to X- linked immune-regulatory gene factor contributing the host's vulnerability to infections in males[80] (Table 1&2).

In present study, the percentage of culture positive cases in LBW neonates(67%) was higher than in normal birth weight neonates. This is

due to the infection rate which is inversely related to the birth weight of newborn and low immunoglobulin G levels and impaired cellular immunity. This similar results found in Barbara Stoll et al study[81](Table2).Percentage of preterm babies in culture proven sepsis is 66%. which is higher than term babies (44%) in culture proven sepsis This is due to inherent deficiencies of both cellular and humoral immunity in preterm infants. According to Barbara J. Stoll et al., study, Incidence of septicemia is inversely proportional to the gestational age of the neonates [81] .In this study ,out of 148neonates with risk of sepsis, 53 (35.18%)had culture proven sepsis and 95 (65.18%) neonates were culture negative sepsis.The predominant organism found in blood culture was acinetobacter and followed by klebsiella.Acinetobacter (gram negative bacteria)is a one of the emerging potential pathogen in neonatal septicemia which was frequently isolated in the recent years .[82,83,84] .Table5 &graph 3 describes the clinical parameters in culture proven sepsis

In culture proven sepsis, the decreasing order of percentage of each clinical parameter are given below. Poor feeding 88%,lethargy 79%, temp instability 71%,apgarscore <7 (67%),oxygen needed babies (62%) which are statistically very much significant with culture proven sepsis,(p value <0.0001) .So health care providers give more attention to babies whose present with these clinical parameters .Except birth asphyxia all other parameters like Positive x ray findings, CRT >3,MSAF,PROM are

statistically considerably significant with culture proven sepsis(p value is <0.05)[84,85]

UTILITY OF CRP

A single value of CRP is not enough to diagnose neonatal septicaemia. It is because CRP value is increased in infection and also physiological conditions like Intraventricular hemorrhage, stressful delivery, fetal distress, meconium aspiration and perinatal asphyxia.

In later conditions the CRP levels usually come back to normal within 24–48 hours but in infective condition it remains high even after 48 hours.So measurement of CRP after 48 hours is required for enhancing the sensitivity and eliminates false positive results. Therefore in my study CRP was evaluated for two times, one after 6 hrs of clinical suspicion of sepsis (CRP1.).Second one after 48 hours of clinical suspicion sepsis (CRP2).

Cut of point for measurement of CRP >5mg/L was used in my study. In culture proven sepsis both CRP 1 and CRP 2 showed high sensitivity (83 %,96%)and high NPV(82.3%,95%) with significant p value <0.001 compared with culture negative sepsis.This results is most consistent with Pal et al., Jadhav et al, Chacha et al results which shows that CRP is a sensitive indicator in diagnosing neonatal sepsis[86,87,88].Among two CRP measurements CRP2 has high sensitivity and NPV than CRP1. So CRP 2 is a good sensitive indicator than CRP1.This result is similar with results of other studies.[89]Table(6,7 and 12)

UTILITY OF CELLULAR PARAMETERS

In culture proven sepsis using cut of point >0.2 for I/T ratio has high specificity (94.73%) and high PPV(54.5%) compared with culture negative sepsis and also have significant p value 0.0003. This result is concordant with that of Ghosh et al[90] and other studies [91].This study revealed I/T ratio is a specific marker for diagnosing neonatal sepsis.(table 10).

In culture proven sepsis, next to I/T ratio, ANC using cut of value <1800/µL showed high specificity (92%) but low sensitivity (1.92%) which was not significant when compared with culture negative sepsis (p value=0.6.) The reason for low sensitivity is due to low number of positive neutropenia cases, age of neonates, blood sampling , the severity of infection .These results were more consistent with study reports of Ghosh et al and Jadhave et al.[87,90] (table 9)

In culture proven sepsis abnormal WBC count (cut off point <5000 or >15000/µL) has high NPV (71.4%) next to CRP. The percentage of sensitivity,specificity, PPV of WBC are 62.2%,52.63%,42.3% respectively. But comparison between proven sepsis and culture negative sepsis showed p value =0.1 which is not significant. These results were more consistent with Basu et al and Buch et al. These variations reported by the different authors may be due to different blood sampling time and age of neonates.(table 8).

Platelet count using cut of point <1.5 lakhs showed 83.1% specificity and low sensitivity (17%),PPV and NPV percentage are 33.3% and 63.7% respectively which is consistent with Ghosh et al reports. But comparison of platelet count between culture proven sepsis and culture negative sepsis is not significant (pvalue is 0.9)[90](table 11)

CRP AND CELLULAR PARAMETERS (table 13,14 15 16))

Positive I/T ratio (>0.2)and WBC counts (<5000 or >15000) were statistically more significant with CRP -1 positive result (pvalue=0.03 for I/T ratio,pvalue <0.0001for WBC)

Positive WBC counts and platelet counts were statistically more significant with CRP -2 positive results (p<0.0001 for WBC and <0.05 for platelet).

Among all measured cellular parameters only I/T ratio has significant correlation with both culture proven sepsis and CRP.This result was most consistent with other study[91] (Table 17,18,19,20)

CLINICAL PARAMETERS WITH CRP1 AND CRP2 (Table 21&22)

1.Comparison of Clinical parameters with CRP1:

Neonates presented with symptom of lethargy shows more statistical significance (p value <0.0001) with CRP1

Other clinical parameters like Temperature instability, Assisted delivery, prematurity ,Low birth weight ,positive X ray finding shows considerable statistical, significance with CRP1 (p value <0.05).

CRT, oxygen requirement ,Apgar <7 ,PROM, male gender,Birth asphyxia , MSAF are not statistically significant with CRP1.

2.Comparison of clinical parameters with CRP2:

Prematurity, lethargy, poor feeding, are statistically more significant with CRP2 (p value <0.0001)

Temperature instability, CRT>3, Low Apgar score<7, assisted delivery, LBW, prematurity, positive xray finding are moderately significant with CRP2 (p value <0.05).

Oxygen requirement, PROM,birth asphyxia, MSAF are not statistically significant with CRP2.

Comparison of clinical parameters with both CRP and culture proven sepsis(table 5,21,22)

Neonates presenting with above mentioned risk factors ,clinical signs and symptoms are statistically significant with both CRP 1& 2 and culture proven sepsis except neonates who presented with history of PROM, MSAF,Birth asphyxia ,requirement oxygen in respiratory distress. Any neonate having elevated CRP with clinical presentation of any of the above risk factors,health care providers should pay more attention with these

babies and start immediate treatment for good prognosis.Preterm ,Low birth weight babies presented with clinical features of lethargy,poor feeding temperature instability and low Apgar score <7along with elevated CRP should require close observation and immediate treatment.

SUMMARY

Study of Evaluation of CRP in neonatal sepsis in comparison with cellular and clinical parameters was conducted in our institution during the period of feb2017-july 2017.Totally 148 neonates with risk of sepsis were included in my study After getting informed consent from parents/guardian, clinical data of all neonates were entered into study proforma . Then measurement of CRP (CRP1,CRP2),cellular parameters (WBC,ANC ,I/T ratio, Platelet count)and Blood culture was done in all neonates with risk of sepsis. Based on blood culture results neonates were classified into culture positive sepsis and culture negative sepsis. Comparison of CRP, cellular parameters and clinical parameters with culture proven and culture negative sepsis was done .Comparison of cellular parameters with CRP positive and negative cases was done. Comparison of CRP1 and CRP2 with clinical parameters was done. To conclude CRP is best sensitive marker which can be used in early diagnosis of neonatal sepsis .CRP measurement after 48 hours enhance the sensitivity and NPV. I/T ratio is a most specific marker that can be used in early diagnosis of neonatal sepsis along with CRP. Elevated CRP along with clinical signs of poor feeding, lethargy .temperature instability ,low Apgar score should be considered promptly for early treatment to reduce neonatal mortality and morbidity.

CONCLUSION

In my study CRP assay has high sensitivity and NPV for diagnosing neonatal sepsis. CRP assay after 48 hours of clinical suspicion of sepsis enhance the sensitivity and NPV in neonatal sepsis. Among the Cellular parameter I/T ratio has high specificity and PPV in diagnosing neonatal sepsis.

In developing countries like India where blood culture is limited, neonates with elevated CRP and raised I/T ratio together with clinical symptoms and signs of poor feeding,lethargy , temperature instability and low Apgar score <7should be observed closely and need appropriate sepsis management which helps to reduce morbidity and mortality.Based on these findings ,CRP assay and measurement of I/T ratio can be used as quick,simple economical methods to diagnose neonatal sepsis in developing countries to minimize the duration of antibiotic treatment which can prevent neonates from the microorganisms with emerging antibiotic resistance.

LIMITATION

The main limitation of this study was the small sample size.We did not include other cultures like CSF, Urine, and Surface culture to determine sepsis. CRP and cellular parameters is not evaluated in non infected (normal neonate) because of ethical issues.

FUTURE PERSPECTIVE

The findings of this study are only suggestive .A large longitudinal study may be required to compare CRP, cellular parameters between non infected (normal) neonates and clinically suspected sepsis neonates.

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ANNEXURES

MASTER CHART

ID	MATURITY	SEX	GA	DOB	DOA	MOD	CRY	ACTIVITY	COLOUR	HR	RR	APGAR SCORE1	APGAR SCORE2	BW	BW CLASSI	TEMP	CRT	O2SAT	SUCKING	LETHARGY	ABD DIS	CULTURE	MICRO ORGANISM	CRP1 mg/l	CRP2 mg/l	WBC COUNT /	PLATELET COUNT /	ANC / µl	IG%	IG/TC	CHF	CHF	MOTHER HISTORY
132	TERM	MALE	AGA	4-Jun	4-Jun	ELSCS	NO	ACTIVE	CYANOSED	150	62	3	6	2.3	NORMAL	35.8	<3	97	NORMAL	YES	NO	NEGATIVE		0.1	2	35000	2.2	11480	2.8	0.085	1	2	post dated
72	TERM	FEMALE	AGA	15-May	18-May	ELSCS	YES	ACTIVE	PINK	150	65	8	9	2.46	LBW	35.5	<3	98	NORMAL	NO	NO	NEGATIVE		0.3	13.5	15400	1.61	10518.2	4.3	0.063	2	5	
128	TERM	MALE	AGA	4-Jun	4-Jun	NVD	FEEBLE	ACTIVE	PINK	148	46	6	7	2.66	NORMAL	36.5	<3	96	NORMAL	NO	YES	NEGATIVE		0.3	1	33800	1.88	21226.4	0.8	0.013	2		prom
83	TERM	MALE	AGA	19-May	20-May	ELSCS	YES	ACTIVE	PINK	146	48	7	8	3.64	NORMAL	34.5	<3	99	POOR	YES	YES	NEGATIVE		0.4	0.5	6000	2.52	3696	1.6	0.026	2		
3	LATE PRE	FEMALE	AGA	19-Feb	19-Feb	ELSCS	NO	DULL	PINK	140	42	6	7	2.22	LBW	35	>3	88	POOR	YES	NO	NEGATIVE		0.5	1.2	19000	3.68	11450	2	0.033	7		DM
69	EARLY PRE	FEMALE	AGA	14-May	14-May	NVD	YES	ACTIVE	PINK	150	58	7	8	1.93	LBW	36.1	<3	98	NORMAL	NO	NO	NEGATIVE		0.5	11.3	17200	2.59	9030	1.5	0.029	2		oligohyd
59	EARLY PRE	FEMALE	AGA	12-May	18-May	LSCS	FEEBLE	DULL	PERI CYANOSIS	149	64	5	4	1.7	LBW	34	<3	97	NORMAL	YES	NO	NEGATIVE		0.7	8.1	17400	3.2	5200	4.5	0.114	2		
77	TERM	MALE	AGA	18-May	18-May	NVD	YES	ACTIVE	PINK	150	60	7	8	2.68	NORMAL	36.4	<3	97	NORMAL	NO	NO	NEGATIVE		0.7	15.3	21400	2.23	12989.8	0.7	0.012	2		
93	TERM	FEMALE	AGA	20-May	23-May	ELSCS	YES	DULL	PINK	148	56	7	8	3.2	NORMAL	37	<3	98	NORMAL	YES	NO	NEGATIVE		0.8	2	7700	1.1	3611.3	0.9	0.019	3	4	
124	LATE PRE	MALE	SGA	22-May	22-May	ELSCS	YES	ACTIVE	PERI CYANOSIS	142	56	7	8	1.65	LBW	36.6	<3	98	NORMAL	YES	NO	NEGATIVE		0.8	20.9	4500	1.24	3186	1.8	0.025	5	2	pih
54	EARLY PRE	MALE	AGA	2/20/2017	2/20/2017	NVD	YES	ACTIVE	PINK	146	46	7	8	1.665	LBW	35	<3	96	POOR	YES	YES	NEGATIVE		0.9	8.6	12260	2.12	7000	2.4	0.040	5	2	DM
54A	EARLY PRE	MALE	AGA	8-May	8-May	NVD	YES	ACTIVE	PINK	146	46	7	8	1.665	lbw	36.6	<3	99	POOR	YES	NO	NEGATIVE		0.9	8.5	5200	2.4	4004	15	0.195	2		
178	EARLY PRE	MALE	AGA	16-Jun	16-Jun	ELSCS	FEEBLE	ACTIVE	PINK	140	65	6	7	1.14	VLBW	34	<3	98	NO	NO	NO	NEGATIVE		1.1	0.9	9000	3.1	3465	3.5	0.091	5	2	
16	TERM	MALE	AGA	15-Apr	15-Apr	LSCS	FEEBLE	ACTIVE	PINK	168	58	5	6	2.5	NORMAL	36.5	<3	98	POOR	YES	NO	NEGATIVE		1.2	1.6	5720	0.71	3120	1.6	0.029	5		
34	EARLY PRE	MALE	SGA	4-May	4-May	NVD	YES	ACTIVE	PINK	130	55	7	8	1.3	VLBW	36.5	>3	98	POOR	NO	NO	NEGATIVE		1.3	1.1	6400	4.24	1920	0.8	0.026	2		twin
160	EARLY PRE	MALE	AGA	12-Jun	12-Jun	NVD	NO	DULL	CYANOSED	140	77	1	3	1.775	VLBW	36.5	>3	96	NO	YES	NO	NEGATIVE		1.4	0.8	19400	2.26	10514.8	7.2	0.133	7	2	
4	TERM	FEMALE	AGA	13-Feb	13-Feb	ELSCS	YES	ACTIVE	PINK	130	65	8	9	3.365	NORMAL	35.6	<3	98	NORMAL	YES	NO	NEGATIVE		1.5	33.7	14700	1.46	9114	2	0.030	2		
150	EARLY PRE	FEMALE	AGA	10-Jun	10-Jun	VACCUM	FEEBLE	DULL	PERI CYANOSIS	140	64	6	7	1.67	LBW	35	<3	97	NORMAL	YES	NO	NEGATIVE		1.5	28.8	18800	1.34	10753.6	3.2	0.056	2		heart dis
166	TERM	MALE	AGA	14-Jun	14-Jun	ELSCS	NO	ACTIVE	PERI CYANOSIS	100	56	6	7	2.7	NORMAL	36.2	<3	97	NORMAL	YES	NO	NEGATIVE		1.5	1.1	23800	3.58	16303	4.5	0.066	2		
92	TERM	FEMALE	AGA	23-May	23-May	NVD	YES	DULL	PERI CYANOSIS	160	62	5	6	1.215	VLBW	37	<3	96	NORMAL	YES	NO	NEGATIVE		1.6	6.8	4400	2.16	1174.8	3.7	0.139	1		
126	TERM	MALE	AGA	31-May	31-May	NVD	FEEBLE	DULL	PERI CYANOSIS	150	46	6	6	2.8	NORMAL	36.5	<3	97	NORMAL	NO	NO	NEGATIVE		1.9	2.2	24000	2.07	12960	4	0.074	10		
173	LATE PRE	MALE	SGA	13-Jun	13-Jun	NVD	YES	ACTIVE	PINK	140	54	7	8	2.1	LBW	35.9	<3	98	NORMAL	NO	YES	NEGATIVE		2.1	6.2	36000	0.92	27828	1.3	0.017	12		
14	EARLY PRE	FEMALE	SGA	14-Mar	14-Mar	LSCS	FEEBLE	DULL	PERI CYANOSIS	156	50	4	5	1.17	VLBW	34.5	<3	98	POOR	YES	NO	NEGATIVE		2.2	4	4400	0.64	2490	0.5	0.009	2		
152	TERM	FEMALE	AGA	11-Jun	11-Jun	NVD	YES	ACTIVE	PINK	140	64	7	8	2.338	LBW	36.3	<3	98	NORMAL	NO	YES	NEGATIVE		2.4	4.2	15000	1.16	10470	0.8	0.011	2	1	
105 A	TERM	MALE	AGA	11-May	11-May	VACCUM	NO	DULL	CYANOSED	100	20	4	5	2.8	NORMAL	34	>3	96	NORMAL	YES	YES	NEGATIVE		2.4	11.2	45900	3.62	24143.4	4.6	0.087	7	1	dm
5	EARLY PRE	FEMALE	AGA	4-Feb	4-Feb	ELSCS	FEEBLE	DULL	PERI CYANOSIS	130	64	2	4	1.38	VLBW	36	<3	92	POOR	YES	NO	NEGATIVE		2.5	5.6	13800	1.79	7040	0.5	0.009	2		pre ec
13	TERM	MALE	AGA	24-Apr	24-Apr	ELSCS	NO	DULL	PERI CYANOSIS	150	70	4	6	2.5	NORMAL	35.5	<3	97	NORMAL	NO	NO	NEGATIVE		4.4	5.2	12000	2.6	5800	1.7	0.041	2		f ind
19	TERM	MALE	AGA	15-Apr	15-Apr	ELSCS	FEEBLE	DULL	PERI CYANOSIS	146	62	5	6	2.8	LBW	36.5	<3	96	NO	YES	NO	NEGATIVE		4.5	3.2	20180	3.13	10100	0.4	0.007	5		DM
129	TERM	FEMALE	AGA	5-Jun	5-Jun	ELSCS	NO	NO	CYANOSED	120	26	2	3	2.7	NORMAL	35	>3	97	NO	YES	YES	NEGATIVE		5	2.8	6100	3.55	3318.4	2.4	0.044	1	2	abr pla
21	EARLY PRE	MALE	AGA	16-Apr	21-Apr	ELSCS	FEEBLE	DULL	ICTERUS	146	54	4	5	1.5	VLBW	34	<3	98	NO	YES	NO	NEGATIVE		5.2	14.5	16000	2.44	8600	4.5	0.081			
98	TERM	FEMALE	AGA	20-May	23-May	NVD	YES	ACTIVE	ICTERUS	140	40	7	8	2.85	NORMAL	36.2	<3	98	NORMAL	NO	NO	NEGATIVE		5.2	3	13900	2.8	4614.8	3.2	0.096	1		
134	TERM	FEMALE	SGA	3-Jun	3-Jun	NVD	NO	DULL	PINK	150	40	5	6	1.91	LBW	35.6	<3	97	POOR	YES	NO	NEGATIVE		5.2	6.6	6800	4.52	4896	2	0.028	7	2	
168	EARLY PRE	MALE	SGA	13-Jun	13-Jun	NVD	NO	NO	CYANOSED	120	40	1	3	1.24	VLBW	34.1	>3	94	NO	YES	YES	NEGATIVE		5.3	4.8	10700	0.82	2161.4	1.2	0.059		2	
37	TERM	FEMALE	AGA	5-May	5-May	NVD	YES	ACTIVE	PINK	146	46	5	6	3.2	LBW	35.8	<3	97	NORMAL	YES	NO	NEGATIVE		5.5	19.2	19900	2.04	12380	4.7	0.070			dm
31	TERM	MALE	AGA	25-Apr	25-Apr	ELSCS	YES	ACTIVE	PINK	160	50	7	8	2.29	NORMAL	35	<3	97	POOR	YES	NO	NEGATIVE		5.8	29	24400	1.5	1750	0.5	0.009	6		
94	TERM	MALE	AGA	24-May	24-May	NVD	NO	DULL	CYANOSED	140	30	2	4	2.45	LBW	36.5	<3	96	POOR	YES	YES	NEGATIVE		6	60.1	29500	2.58	19470	25	0.379	2	5	
96	EARLY PRE	MALE	SGA	21-May	21-May	LSCS	YES	ACTIVE	PERI CYANOSIS	150	60	7	8	1.545	LBW	35.6	<3	98	NORMAL	NO	NO	NEGATIVE		6	10	17700	1.72	10761.6	6.8	0.112	1	4	
116	TERM	MALE	AGA	29-May	29-May	NVD	YES	DULL	CYANOSED	150	56	7	8	2.53	NORMAL	33	>3	98	POOR	YES	NO	NEGATIVE		6	5.3	10000	3.2	3650	1.5	0.041	6	2	pih
24	EARLY PRE	MALE	AGA	17-Apr	21-Apr	ELSCS	FEEBLE	DULL	PERI CYANOSIS	168	62	5	6	1.3	VLBW	36	<3	99	NO	YES	NO	NEGATIVE		7.1	5.5	12390	2.53	4950	1.5	0.036	2		
24A	EARLY PRE	FEMALE	SGA	1-May	1-May	ELSCS	NO	DULL	PERI CYANOSIS	140	66	5	6	1.23	VLBW	34.1	<3	97	NO	YES	YES	NEGATIVE		7.1	5.5	12390	2.54	4950	1.5	0.036	5	6	
32	TERM	FEMALE	AGA	3-May	3-May	LSCS	YES	DULL	PINK	158	62	7	8	2.845	NORMAL	35	>3	98	NORMAL	YES	NO	NEGATIVE		7.2	7	14700	3.4	9820	1.3	0.019	16		CPD
189	TERM	MALE	AGA	18-Jun	21-Jun	NVD	YES	ACTIVE	PINK	140	33	7	8	3.09	NORMAL	38	<3	96	POOR	YES	NO	NEGATIVE		7.3	10.2	4900	2.5	3567.2	4.8	0.066			
109	TERM	FEMALE	AGA	29-May	29-May	LSCS	YES	ACTIVE	PINK	140	50	7	8	2.71	NORMAL	36.4	<3	98	POOR	YES	NO	NEGATIVE		7.5	6	17200	0.71	11627.2	1.6	0.024	2		prom>18,oligo
133	EARLY PRE	MALE	AGA	26-May	26-May	ELSCS	YES	ACTIVE	PINK	146	40	7	8	1.42	LBW	36.5	<3	98	POOR	YES	NO	NEGATIVE		7.7	0.6	11000	1.57	6314	6.4	0.111			oligohyd

48	EARLY PRE	MALE	SGA	15-May	15-May	LSCS	YES	ACTIVE	PINK	150	48	6	7	1.2	LBW	34	>3	98	POOR	NO	YES	NEGATIVE		8	11.2	10068	0.45	5670	3.9	0.069	2					
151	LATE PRE	MALE	AGA	11-Jun	11-Jun	NVD	FEEBLE	DULL	PINK	140	56	7	8	2.485	LBW	34.6	<3	97	POOR	YES	NO	NEGATIVE		8.3	7.1	13400	3.24	9447	11.5	0.163	5					
35	EARLY PRE	MALE	SGA	4-May	4-May	NVD	YES	DULL	PINK	150	55	7	8	1.245	VLBW	35	<3	98	POOR	YES	NO	NEGATIVE		9.1	1.4	9420	2.4	6400	1.5	0.032						twin
7	EARLY PRE	MALE	AGA	16-Feb	16-Feb	VACCUM	FEEBLE	DULL	PERI CYANOSIS	150	56	6	7	1.8	VLBW	36.5	<3	98	POOR	YES	NO	NEGATIVE		9.5	10.4	23660	2.46	12910	1.4	0.023	5					no
111	LATE PRE	FEMALE	AGA	27-May	27-May	LSCS	YES	ACTIVE	PINK	160	62	6	7	2.8	NORMAL	36.5	<3	96	NORMAL	NO	YES	NEGATIVE		9.6	13.4	11300	1.8	3955	5	0.143	16	6				
127	TERM	MALE	AGA	4-Jun	6-Jun	LSCS	YES	ACTIVE	PINK	140	48	7	8	2.825	NORMAL	40	<3	97	NORMAL	YES	NO	NEGATIVE		9.7	8.6	14300	3.3	10296	5	0.069						
113	EARLY PRE	MALE	AGA	28-May	28-May	NVD	YES	DULL	ICTERUS	150	54	5	7	1.98	LBW	36	<3	98	POOR	YES	NO	NEGATIVE		10.1	10	10300	1.63	4202.4	1.8	0.044						dm
78	TERM	MALE	SGA	19-May	19-May	NVD	YES	ACTIVE	PINK	148	46	7	8	2.08	NORMAL	36.4	<3	97	NORMAL	NO	YES	NEGATIVE		10.2	10.7	17490	3.9	12662.76	1.4	0.019		2				
95	TERM	MALE	AGA	19-May	19-May	NVD	NO	DULL	PERI CYANOSIS	160	52	4	6	2.6	NORMAL	35.2	<3	98	POOR	YES	NO	NEGATIVE		10.5	6	21500	1.73	16254	4.6	0.061	5	7	rh neg			
187	EARLY PRE	MALE	AGA	20-Jun	20-Jun	NVD	FEEBLE	ACTIVE	PERI CYANOSIS	150	60	6	7	1.58	LBW	36	<3	97	NORMAL	NO	NO	NEGATIVE		10.5	11.4	8900	2.09	3168.4	5.6	0.157	2	6	anemia			
142A	EARLY PRE	MALE	SGA	9-Jun	9-Jun	ELSCS	YES	ACTIVE	PINK	140	50	7	8	1.68	LBW	39.1	<3	98	POOR	YES	NO	NEGATIVE		11.2	8.6	11000	1.2	7920	7	0.097	2		prom			
120	EARLY PRE	MALE	SGA	4-Jun	4-Jun	ELSCS	FEEBLE	DULL	PINK	140	64	6	7	0.83	VLBW	36.5	<3	96	POOR	YES	NO	NEGATIVE		11.6	17.1	5100	2.512	1897.2	7.2	0.194	2		pih			
141	TERM	FEMALE	SGA	7-Jun	7-Jun	ELSCS	YES	ACTIVE	PINK	140	36	7	8	2.145	LBW	35.6	<3	98	POOR	YES	NO	NEGATIVE		11.8	10	20200	2.73	15251	7.5	0.099	2		prom,ane			
75	EARLY PRE	MALE	SGA	16-May	16-May	ELSCS	NO	DULL	PERI CYANOSIS	140	58	5	6	1.9	LBW	35.9	<3	98	POOR	YES	NO	NEGATIVE		12.3	10	14200	1.92	8491.6	2.8	0.047	2	5	oligohyd			
89	TERM	MALE	AGA	22-May	28-May	LSCS	NO	DULL	PERI CYANOSIS	150	48	6	7	2.6	NORMAL	35.6	<3	98	POOR	YES	NO	NEGATIVE		12.6	15	27000	2	18765	2.5	0.036	2					
140	EARLY PRE	FEMALE	AGA	2-Jun	2-Jun	ELSCS	YES	ACTIVE	PINK	150	64	6	7	2.145	LBW	36.5	<3	98	POOR	YES	YES	NEGATIVE		12.6	1.5	19500	1.02	9009	2.2	0.048	5		dm			
55	TERM	MALE	AGA	5/23/2017	10-May	NVD	YES	DULL	PERI CYANOSIS	150	50	7	8	3.37	NORMAL	36.5	<3	97	NORMAL	YES	YES	NEGATIVE		12.7	13	6500	2.93	2670	1.2	0.029		6				
135	TERM	FEMALE	AGA	7-Jun	7-Jun	ELSCS	YES	ACTIVE	PINK	140	36	8	9	2.22	LBW	36	<3	98	POOR	YES	NO	NEGATIVE		12.9	8.9	12800	2.09	9664	1.5	0.020	2		prom,oligo			
105	TERM	MALE	AGA	26-May	28-May	NVD	YES	ACTIVE	PINK	158	56	6	7	3.2	NORMAL	36.8	<3	97	NORMAL	NO	YES	NEGATIVE		13.7	15	16000	3.37	14560	10	0.110	2					
50	LATE PRE	FEMALE	AGA	6-May	6-May	LSCS	YES	ACTIVE	PINK	150	66	7	8	1.87	NORMAL	35.4	<3	97	NORMAL	NO	YES	NEGATIVE		15	2.8	21600	1.6	12120	0.2	0.005	7	5				
56	TERM	MALE	AGA	9-May	9-May	NVD	NO	DULL	PERI CYANOSIS	140	65	5	6	2.6	NORMAL	38	<3	97	NORMAL	YES	NO	NEGATIVE		15.2	13.2	26000	2.67	19200	1	0.014	2					
106	TERM	MALE	AGA	25-Mar	25-Mar	ELSCS	NO	DULL	PINK	140	64	5	6	2.96	NORMAL	36.5	<3	96	NORMAL	NO	NO	NEGATIVE		15.8	21.5	14500	1.61	5452	7.6	0.202	2		fetal dis			
149	LATE PRE	MALE	SGA	10-Jun	10-Jun	NVD	FEEBLE	DULL	PERI CYANOSIS	168	56	7	8	2.025	LBW	35.6	>3	97	POOR	YES	NO	NEGATIVE		16.2	6.2	18700	2.66	11220	5	0.083	2	4				
156	TERM	MALE	AGA	6-Jun	6-Jun	ELSCS	YES	ACTIVE	PINK	150	44	8	9	2.73	NORMAL	35	<3	98	POOR	YES	YES	NEGATIVE		16.8	12.6	4000	3.5	2708	4.7	0.069	2	1	dm,ane,hbv			
82	TERM	FEMALE	LGA	17-May	17-May	LSCS	NO	DULL	PERI CYANOSIS	160	64	3	7	3.5	NORMAL	35	<3	97	POOR	NO	NO	NEGATIVE		18.1	24	10900	1.89	7390.2	1.8	0.027	1	1				
42	LATE PRE	MALE	AGA	6-May	6-May	NVD	NO	DULL	PINK	148	61	5	7	2.945	NORMAL	35	>3	96	NORMAL	NO	NO	NEGATIVE		18.2	2.6	29000	3.09	12600	2	0.051						
39	TERM	FEMALE	AGA	8-May	8-May	LSCS	YES	ACTIVE	PINK	150	48	7	8	2.56	NORMAL	35	<3	97	NORMAL	NO	YES	NEGATIVE		18.7	17	28060	3.11	22200	1.6	0.020	1		twin			
101	TERM	MALE	AGA	22-May	28-May	ELSCS	YES	ACTIVE	PINK	160	66	6	7	2.5	NORMAL	37.1	<3	98	NORMAL	NO	NO	NEGATIVE		21	17	14900	1.39	10340.6	6.4	0.092	2	2				
60	TERM	MALE	AGA	12-May	12-May	NVD	YES	DULL	PERI CYANOSIS	150	65	7	8	3.075	NORMAL	36.4	<3	97	NORMAL	YES	NO	NEGATIVE		21.3	69	17000	2.14	10630	2.1	0.033	1					
100	TERM	MALE	AGA	28-May	28-May	ELSCS	YES	ACTIVE	PERI CYANOSIS	150	56	7	8	2.91	NORMAL	36.4	<3	98	POOR	NO	NO	NEGATIVE		22.9	19.2	13100	2.81	7755.2	1.2	0.020	4	2	fetal dis			
87	TERM	MALE	AGA	18-May	22-May	NVD	NO	DULL	PINK	148	52	7	8	2.85	NORMAL	36	<3	98	POOR	YES	NO	NEGATIVE		25	10.8	22500	1.92	14310	0.6	0.009	2					
79	EARLY PRE	MALE	AGA	15-May	15-May	LSCS	YES	ACTIVE	PINK	152	50	7	8	1.865	LBW	37	<3	98	NORMAL	NO	NO	NEGATIVE		28	14.8	14100	2.8	8699.7	0.7	0.011	15	5	PROM			
53	TERM	MALE	AGA	9-May	9-May	LSCS	YES	ACTIVE	PERI CYANOSIS	146	47	7		8	NORMAL	34.5	<3	98	NORMAL	YES	NO	NEGATIVE		29.6	12.5	23200	2.6	12710	1	0.016			dm			
157	TERM	FEMALE	AGA	8-Jun	8-Jun	ELSCS	YES	ACTIVE	PINK	140	54	7	8	3.285	NORMAL	36.5	<3	98	POOR	YES	NO	NEGATIVE		36.6	15.1	23000	2.2	19044	2.8	0.034	1		dm			
52	LATE PRE	MALE	AGA	5/11/2017	5/11/2017	LSCS	YES	ACTIVE	PERI CYANOSIS	140	42	6	7	2.5	NORMAL	38	<3	96	NORMAL	NO	NO	NEGATIVE		46.8	0.8	23700	2.48	18300	0.8	0.010			DM			
65A	TERM	MALE	AGA	13-May	13-May	LSCS	YES	ACTIVE	PINK	150	65	7	8	2.9	NORMAL	36	<3	98	POOR	YES	YES	NEGATIVE		50.2	13.5	15020	1.78	8831.76	2.8	0.048	2		fever			
175	EARLY PRE	MALE	AGA	16-Jun	16-Jun	VACCUM	YES	ACTIVE	PINK	140	64	6	7	1.75	LBW	35.9	<3	98	POOR	NO	NO	NEGATIVE		0.5	5.3	3600	2.51	1879.2	3.2	0.061	9					
183	TERM	MALE	AGA	14-Jun	19-Jun	NVD	YES	ACTIVE	PINK	156	48	7	8	2.9	NORMAL	35.6	<3	98	POOR	YES	NO	NEGATIVE		0.7	1.2	11000	2.14	2266	1.6	0.078	3					
147	TERM	MALE	AGA	7-Jun	7-Jun	LSCS	FEEBLE	DULL	PERI CYANOSIS	140	54	7	8	2.865	NORMAL	37.5	>3	98	POOR	YES	NO	NEGATIVE		1.1	4.8	5900	1.92	2495.7	4.3	0.102	2	4				
12	TERM	FEMALE	AGA	21-Apr	21-Apr	ELSCS	FEEBLE	ACTIVE	PINK	154	62	7	8	2.5	LBW	34.7	<3	97	POOR	NO	NO	NEGATIVE		2.4	23.5	19000	3.2	10360	3.6	0.065		10				
28	LATE PRE	MALE	AGA	1-May	1-May	NVD	YES	ACTIVE	PINK	146	42	8	9	2.215	LBW	36.5	<3	97	POOR	YES	NO	NEGATIVE		2.9	4	9700	1.87	5160	1.2	0.022	6					
122	TERM	MALE	LGA	1-Jun	1-Jun	ELSCS	YES	ACTIVE	PINK	140	30	7	8	4.435	OVER WT	36.6	<3	98	POOR	NO	YES	NEGATIVE		5.8	10.2	22600	2.86	15955.6	6.6	0.093	7		gdm			
33	TERM	MALE	AGA	4-May	4-May	NVD	NO	DULL	CYANOSED	138	68	3	4	3.2	NORMAL	35.6	>3	96	NO	YES	NO	NEGATIVE		6	16.1	12190	2.28	10040	5.8	0.064	7	2				
163	EARLY PRE	MALE	AGA	12-Jun	12-Jun	NVD	NO	DULL	CYANOSED	120	35	1	1	1.1	VLBW	34.2	>3	88	NO	YES	YES	NEGATIVE		6.8	7.1	14000	1.92	3024	1.6	0.074	2	2				
130	EARLY PRE	FEMALE	AGA	6-Jun	9-Jun	NVD	FEEBLE	DULL	PERI CYANOSIS	150	54	5	6	1.4	VLBW	34	<3	97	NORMAL	YES	YES	NEGATIVE		9	8.5	15000	3.42	7650	1	0.020	7					
136	EARLY PRE	FEMALE	AGA	7-Jun	7-Jun	ELSCS	FEEBLE	DULL	CYANOSED	80	60	2	4	1.25	VLBW	34.5	>3	96	NORMAL	YES	YES	NEGATIVE		10.6	12.1	23800	1.94	6140.4	0.8	0.031	2	2	abr pla			

57	TERM	MALE	AGA	8-May	8-May	LSCS	YES	ACTIVE	PINK	142	46	6	7	2.6	NORMAL	36	<3	97	NORMAL	NO	NO	NEGATIVE		15	0.8	2420	0.13	690	15.7	0.210	2				
102	TERM	FEMALE	SGA	27-May	27-May	NVD	YES	ACTIVE	PINK	156	50	7	8	1.8	LBW	36.4	<3	98	NORMAL	NO	NO	NEGATIVE		17.8	20.1	22400	2.86	16643.2	5.3	0.071	2				
6	EARLY PRE	FEMALE	AGA	16-Apr	16-Apr	ELSCS	FEEBLE	ACTIVE	PERI CYANOSIS	156	62	5	7	1.7	LBW	37	>3	95	POOR	YES	NO	NEGATIVE		24	16	20490	3.18	12900	1.4	0.022			hyper.thy		
41	EARLY PRE	FEMALE	SGA	8-May	8-May	LSCS	YES	DULL	PINK	186	58	6	7	1	LBW	34	<3	97	POOR	YES	YES	NEGATIVE		26.3	48.1	16710	1.4	7790	4.2	0.097	7		fever		
74	EARLY PRE	MALE	AGA	16-May	16-May	ELSCS	NO	NO	PERI CYANOSIS	130	58	2	3	1.645	LBW	34	>3	96	POOR	YES	NO	NEGATIVE		65.2	5.4	12090	2.75	4473.3	4	0.108	3		dm/aph		
145	TERM	FEMALE	AGA	6-Jun	10-Jun	LSCS	YES	ACTIVE	PINK	140	40	7	8	2.156	LBW	38.5	<3	98	POOR	YES	NO	POSITIVE	klebsiella oxytoca	0.12	4.7	6200	2.22	4699.6	6.8	0.090	2	4	failed in		
146	EARLY PRE	FEMALE	SGA	6-Jul	15-Jul	ELSCS	YES	DULL	PINK	140	60	7	8	1.5	VLBW	35.4	<3	97	POOR	YES	NO	POSITIVE	klebsiella pnemoniae	2	3.3	5700	1.63	1761.3	1.9	0.061	2	11	plh		
174	LATE PRE	FEMALE	AGA	21-Jul	21-Jul	ELSCS	YES	DULL	PERI CYANOSIS	140	42	8	8	2.515	NORMAL	35.7	<3	95	NO	YES	NO	POSITIVE	acinetobacter	0.225	4.6	22000	1.85	10450	2.5	0.053	6		twin		
131	TERM	MALE	AGA	6-Jun	6-Jun	ELSCS	YES	ACTIVE	PINK	150	48	7	8	3.48	NORMAL	39.8	<3	98	NORMAL	NO	NO	POSITIVE	enterococci	0.4	1.6	19600	2.28	16366	3.5	0.042	2		prom		
153	EARLY PRE	FEMALE	AGA	11-Jul	11-Jul	NVD	FEEBLE	DULL	PINK	140	66	6	7	1.595	LBW	35.6	<3	97	NORMAL	YES	NO	POSITIVE	coagulus negative staphylococc	0.5	2.2	9100	3.54	3094	12	0.353	8				
170	TERM	MALE	AGA	14-Jun	14-Jun	NVD	YES	DULL	PINK	140	42	7	8	2.435	NORMAL	35.7	<3	97	NORMAL	YES	YES	POSITIVE	klebsiella pnemoniae	0.6	4.2	16700	2.63	10504.3	1.9	0.030	5				
67	TERM	FEMALE	AGA	9-May	9-May	NVD	YES	ACTIVE	PINK	156	62	5	6	2.64	NORMAL	36	>3	97	NO	NO	NO	POSITIVE	acinetobacter	0.7	13.6	11740	1.22	6720	1.8	0.031	7	6			
121	TERM	MALE	LGA	5-Jun	5-Jun	ELSCS	FEEBLE	DULL	PERI CYANOSIS	140	20	3	5	3.75	NORMAL	35.6	>3	96	NO	YES	NO	POSITIVE	klebsiella pnemoniae	0.7	33	23000	1.04	6187	0.9	0.033	1	2			
119	TERM	MALE	AGA	3-Jun	3-Jun	NVD	FEEBLE	ACTIVE	PINK	150	60	6	7	3.2	NORMAL	35	<3	97	NORMAL	YES	NO	POSITIVE	pseudomonas	0.8	2.3	18000	2.48	11520	6	0.094	2	2			
117	EARLY PRE	FEMALE	SGA	31-May	31-May	NVD	FEEBLE	DULL	PERI CYANOSIS	140	66	5	6	0.99	VLBW	38	<3	97	NO	YES	NO	POSITIVE	pseudomonas	1.2	6	22800	2.4	3921.6	1.2	0.070	2	5	pre ec		
26	EARLY PRE	MALE	SGA	1-May	1-May	NVD	FEEBLE	DULL	PINK	152	62	5	6	1	VLBW	35	>3	94	POOR	YES	NO	POSITIVE	Ecoli	1.3	3	13000	2.52	2480	1.1	0.055	2		prom		
46	LATE PRE	FEMALE	AGA	9-May	9-May	LSCS	YES	ACTIVE	PERI CYANOSIS	150	52	7	8	2.4	NORMAL	36.2	<3	97	NORMAL	NO	NO	POSITIVE	coagulus negative staphylococc	2.3	5.4	21830	2.24	7830	2	0.053	3				
85	TERM	MALE	SGA	18-May	18-May	ELSCS	YES	ACTIVE	PINK	140	48	7	8	1.97	LBW	35.8		99	POOR	NO	YES	POSITIVE	coagulus negative staphylococc	2.3	5.4	9100	1.53	3230.5	1.5	0.042	6	4	fever		
139	TERM	FEMALE	AGA	4-Jun	7-Jun	NVD	YES	ACTIVE	PINK	152	56	7	8	2.707	NORMAL	38.9	<3	97	POOR	YES	NO	POSITIVE	ECOLI	2.5	1.9	7600	2.04	5836.8	1.8	0.023	2				
123	TERM	MALE	SGA	2-Jun	2-Jun	ELSCS	YES	ACTIVE	PINK	150	44	7	8	1.86	OVER WT	39	<3	97	POOR	NO	YES	POSITIVE	ECOLI	2.6	4	14000	2.39	8764	0.6	0.010	2		prom		
108	TERM	FEMALE	AGA	27-May	27-May	ELSCS	YES	ACTIVE	PINK	146	48	7	8	2.75	NORMAL	37.5	<3	98	POOR	NO	YES	POSITIVE	klebsiella pnemoniae	3.5	5.6	12400	2.48	9585.2	3.3	0.043	2				
171	TERM	FEMALE	AGA	15-Jun	15-Jun	ELSCS	NO	DULL	CYANOSED	140	64	5	6	2.31	LBW	34.5	>3	98	NORMAL	YES	NO	POSITIVE	enterococci	4.5	3	16000	2.97	10016	2.6	0.042	7	1			
43	EARLY PRE	FEMALE	AGA	3-May	3-May	LSCS	NO	DULL	PINK	146	46	5	7	1.805	LBW	36.4	<3	97	NO	NO	YES	POSITIVE	acinetobacter	4.8	15.9	14700	1.6	5010	0.2	0.006	2				
112	TERM	FEMALE	SGA	29-May	29-May	ELSCS	YES	ACTIVE	PINK	140	62	6	7	1.685	LBW	34.5	<3	98	POOR	NO	NO	POSITIVE	klebsiella pnemoniae	4.8	6.8	19600	1.84	9898	2.5	0.050	6		plh		
138	TERM	MALE	AGA	8-Jun	8-Jun	ELSCS	YES	ACTIVE	PINK	140	48	7	8	3.095	NORMAL	39	<3	98	POOR	YES	NO	POSITIVE	Ecoli	5.1	4	17000		13345	3.5	0.045	2		prom		
15	EARLY PRE	MALE	AGA	25-Apr	25-Apr	NVD	FEEBLE	DULL	PINK	154	64	5	7	1.41	VLBW	36	<3	98	NO	YES	NO	POSITIVE	acinetobacter	5.5	8.6	9460	2.53	3338	1.4	0.038	2				
27	EARLY PRE	MALE	AGA	2-May	2-May	ELSCS	YES	ACTIVE	PINK	152	54	7	8	2.08	LBW	35.5	<3	97	NO	NO	NO	POSITIVE	acinetobacter	5.7	5.2	13000	2.8	8450	1.3	0.020	2				
125	EARLY PRE	MALE	SGA	1-Jun	1-Jun	NVD	YES	ACTIVE	PINK	150	52	7	8	1.125	VLBW	34	<3	98	POOR	YES	NO	POSITIVE	klebsiella pnemoniae	5.7	16.2	13400	2.69	7611.2	5.8	0.102	6				
9	TERM	FEMALE	AGA	24-Apr	24-Apr	ELSCS	NO	DULL	PERI CYANOSIS	150	42	3	4	2.8	NORMAL	35	>3	97	NO	YES	NO	POSITIVE	acinetobacter	5.8	1.2	23400	2.66	13370	1.2	0.021	7				
33A	TERM	MALE	AGA	4-May	8-May	ELSCS	YES	ACTIVE	PERI CYANOSIS	146	50	3	8	3.08	NORMAL	36.8	<3	95	POOR	YES	NO	POSITIVE	acinetobacter	6	10	12190	2.2	10400	5.8	0.064	2		f ind		
110	EARLY PRE	MALE	AGA	29-May	29-May	ELSCS	YES	DULL	PINK	140	64	6	7	2.14	LBW	40	<3	97	POOR	YES	NO	POSITIVE	Ecoli	6.2	7.8	11500	1.7	5301.5	6.1	0.132	2		fever		
190	LATE PRE	MALE	AGA	19-Jun	19-Jun	NVD	NO	DULL	PERI CYANOSIS	182	64	7	8	2.45	lbw	36.5	<3	97	POOR	YES	NO	POSITIVE	acinetobacter	6.5	8.8	17100	2.2	10089	1	0.017	2				
169	EARLY PRE	FEMALE	AGA	13-Jun	13-Jun	NVD	YES	ACTIVE	PERI CYANOSIS	146	64	6	7	1.92	LBW	35.8	<3	97	NORMAL	NO	NO	POSITIVE	klebsiella pnemoniae	6.9	5.2	20300	2.64	14697.2	2.4	0.033	2				
23	LATE PRE	FEMALE	AGA	23-Apr	23-Apr	NVD	YES	ACTIVE	PINK	180	70	7	8	2.055	LBW	36.5	<3	97	POOR	YES	NO	POSITIVE	acinetobacter	7.4	6.7	11000	2.39	6200	3.6	0.064	7				
97	LATE PRE	MALE	AGA	24-May	24-May	ELSCS	NO	DULL	PERI CYANOSIS	110	30	1	2	1.9	NORMAL	34.5	>3	95	NO	YES	YES	POSITIVE	klebsiella oxytoca	8.3	10.2	19800	2.5	7147.8	1.1	0.030	2	5	oligohyd		
182	LATE PRE	MALE	AGA	17-Jun	17-Jun	NVD	FEEBLE	DULL	PINK	150	70	8	9	2.5	NORMAL	35.2	<3	96	NO	YES	NO	POSITIVE	acinetobacter	8.7	7	6600	2.51	3326.4	2.4	0.048	16				
165	TERM	FEMALE	AGA	6-Jun	9-Jun	NVD	YES	ACTIVE	PINK	140	42	7	8	3.425	NORMAL	38.2	<3	98	POOR	YES	NO	POSITIVE	klebsiella pnemoniae	8.9	10.1	9400	2.84	8610.4	4.6	0.050	16				
130	TERM	MALE	AGA	3-Jun	5-Jun	NVD	YES	ACTIVE	PINK	150	56	7	8	3.345	NORMAL	40	<3	98	NORMAL	YES	NO	POSITIVE	acinetobacter	9	11.2	7300	2.1	5110	11	0.157	2				
99	EARLY PRE	MALE	AGA	26-May	26-May	NVD	YES	DULL	PERI CYANOSIS	150	60	7	8	1.145	VLBW	36.5	<3	98	NORMAL	YES	NO	POSITIVE	acinetobacter	9.5	16.5	5600	3.2	3808	11	0.162					
144	TERM	FEMALE	AGA	17-Jun	17-Jun	ELSCS	YES	ACTIVE	PINK	160	48	8	9	3.3	NORMAL	35.4	<3	98	POOR	YES	NO	POSITIVE	klebsiella pnemoniae	9.6	15	18000	3.9	10350	1.5	0.026	5		mat fever,prom		
104	EARLY PRE	MALE	AGA	27-May	27-May	NVD	FEEBLE	DULL	PINK	150	60	7	8	1.393	VLBW	36.6	<3	98	NORMAL	YES	YES	POSITIVE	acinetobacter	9.8	25.5	10600	2.14	3900.8	6.8	0.185	2	6	dm		
107	EARLY PRE	MALE	AGA	25-May	25-May	NVD	YES	ACTIVE	PERI CYANOSIS	150	56	7	8	1.405	VLBW	35.3	<3	97	POOR	NO	NO	POSITIVE	staphylococcus aureus	9.8	25.5	10600	2.14	4261.2	10.2	0.254	5		dm		
68	EARLY PRE	FEMALE	SGA	12-May	12-May	NVD	NO	DULL	PERI CYANOSIS	148	59	5	6	1.9	LBW	35	<3	96	NORMAL	YES	NO	POSITIVE	citrobactor	10	15.5	6520	0.18	2680	6.9	0.144					
114	TERM	FEMALE	AGA	28-May	31-May	NVD	YES	DULL	PINK	150	48	6	7	2.83	NORMAL	38.1	<3	97	POOR	YES	YES	POSITIVE	acinetobacter	11.5	10.2	10900	3.81	3684.2	1.8	0.053	3				
115	TERM	MALE	AGA	27-May	27-May	ELSCS	YES	DULL	PINK	140	48	7	8	2.645	NORMAL	35.6	<3	97	POOR	NO	YES	POSITIVE	acinetobacter	12	11.8	20000	2.44	14440	2.2	0.030	4	4	dm		
58	TERM	FEMALE	AGA	11-May	11-May	ELSCS	FEEBLE	DULL	PINK	158	60	6	7	2.7	NORMAL	35.7	<3	98	NO	NO	YES	POSITIVE	klebsiella pnemoniae	12.5	12.3	29140	2.54	13250	8.3	0.156	2		plh		

162	TERM	FEMALE	AGA	12-Jul	12-Jul	NVD	FEEBLE	DULL	PERI CYANOSIS	150	67	6	7	3.105	NORMAL	36.5	>3	97	NORMAL	YES	NO	POSITIVE	acinetobacter	12.9	10.1	19100	1.56	13198.1	1.1	0.016	1		
118	TERM	FEMALE	SGA	2-Jun	2-Jun	NVD	YES	ACTIVE	PINK	140	64	6	7	2.045	NORMAL	37	<3	98	NORMAL	NO	NO	POSITIVE	klebsiella oxytoca	13	9.6	16900	1.42	12759.5	3.5	0.046	2	2	
176	TERM	MALE	AGA	15-Jun	16-Jun	FORCEPS	NO	DULL	PERI CYANOSIS	140	64	5	6	3.18	NORMAL	37	<3	97	NO	YES	NO	POSITIVE	coagulus negative staphylococc	15.1	58.2	12400	2.06	9424	6	0.079	7		
51	LATE PRE	MALE	AGA	5/10/2017	5/10/2017	LSCS	YES	ACTIVE	PINK	148	48	7	8	2	LBW	36	<3	96	NORMAL	NO	YES	POSITIVE	acinetobacter	16.8	15	12880	2.99	6600	1.1	0.021	2	2	
36	EARLY PRE	FEMALE	AGA	3-May	3-May	ELSCS	YES	DULL	PINK	148	48	6	7	1.43	VLBW	36	<3	98	NO	YES	YES	POSITIVE	staphylococcus aureus	17.9	5.9	22000	3.2	15378	4.9	0.070	2		dm
47A	LATE PRE	FEMALE	SGA	11-May	11-May	ELSCS	YES	DULL	PINK	156	64	5	6	1.65	LBW	35.8	<3	97	NO	YES	NO	POSITIVE	pseudomonas	18	20.4	20000	1.45	9860	1.9	0.041	5	5	fever
60A	LATE PRE	MALE	SGA	11-May	11-May	ELSCS	YES	ACTIVE	PINK	140	64	6	7	1.65	LBW	35.6	<3	97	NO	YES	YES	POSITIVE	pseudomonas	19.2	21	19670	3.35	15972	14.2	0.175	2		fever
64	EARLY PRE	MALE	SGA	12-May	12-May	NVD	YES	ACTIVE	PINK	150	55	7	8	1.98	LBW	34	<3	97	POOR	NO	NO	POSITIVE	acinetobacter	19.3	12.3	14500	3.12	7177	4.5	0.091	2		dm
41	EARLY PRE	FEMALE	SGA	4-May	4-May	NVD	YES	ACTIVE	PINK	160	60	4	5	1	LBW	34.8	<3	97	NO	NO	NO	POSITIVE	acinetobacter	26.3	48.1	10710	1.4	5237	9.9	0.202	16		
79	EARLY PRE	MALE	AGA	15-May	15-May	LSCS	YES	ACTIVE	PINK	152	50	7	8	1.865	lbw	35.4	<3	97	POOR	YES	NO	POSITIVE	acinetobacter	28	14.8	8800	3.4	3854.4	6.8	0.155	3		prom
76	TERM	MALE	AGA	17-May	17-May	NVD	YES	DULL	PINK	150	50	7	8	2.815	NORMAL	36.2	<3	97	NORMAL	NO	NO	POSITIVE	acinetobacter	28.8	15	21000	1.69	12621	6.1	0.101	2	4	pph
44	LATE PRE	FEMALE	AGA	4-May	4-May	NVD	YES	ACTIVE	PINK	146	48	7	8	2.175	LBW	36.6	<3	96	NO	NO	NO	POSITIVE	acinetobacter	118.4	145	16710	1.9	10190	2.4	0.038	1		

1.MSAF-MECONIUM STAINEF AMNIOTIC FLUID
6 .X RAY ABDOMEN -POSITIVE
7.BIRTH ASPHYXIA
10.PROM
5.XRAY ABDOMEN-POSITIVE

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Evaluation of C reactive Protein (CRP) in Neonatal Sepsis in comparison with Cellular and clinical parameters.

Principal Investigator : Dr. V Gomathi

Designation : PG, MD (Biochemistry)

Department : Department of Biochemistry
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 24.02.2017 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY, 5/4/17
IEC, SMC, CHENNAI.

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

NEONATAL CASE RECORD SHEET

SNCU Reg. No.....

Doctor in charge ..... MCTS No. | | | | | | | | | | | | | | | | | |

Baby of (Mother's Name)			Sex : Male / Female / Ambiguous
Father's Name			Category: General / OBC / SC / ST
Complete Address with Village Name / Ward No.			
Contact No. & Relation	1.	2.	
Date and Time of Birth	...../...../20.....	Birth Weight (Kg) :	
Date and Time of Admission	...../...../20.....	Age on Admission :	Wt. on Admission (Kg) :
Date and Time of Discharge	...../...../20.....	Age on Discharge :	Wt. on Discharge (Kg) :
Type of Admission	Inborn / Out born (Health Facility Referred) / Out Born (Community Referred)		
Place of Delivery	Home / Ambulance / Pvt. Hospital / Govt. Hospital (Name) :		
Referred From		Mode of Transport : Self Arranged / Govt. Provided	

Indication for Admission (Encircle the most relevant single indication, If multiple indication also mention all relevant numbers in the end as per priority)

1. Permaturity <34 weeks
2. Low Birth Weight <1800 gm.
3. Perinatal Asphyxia
4. Neonatal Jaundice
5. Resp. Distress (Rate>60 or Grunt / Retractions)
6. Large Baby (>4 Kg. at 40 weeks)
7. Refusal to Feed
8. Central Cyanosis
9. Apnea / Gasping
10. Neonatal Convulsions
11. Baby of Diabetic mother
12. Oliguria
13. Abdominal Distension
14. Hypothermia <35.4 °C
15. Hyperthermia >37.5 °C
16. Hypoglycemia <45 mg%
17. Shock : Cold Periphery with CFT >3 sec & Weak Fast Pulse
18. Meconium Aspiration
19. Bleeding
20. Diarrhoea
21. Major Congenital Malformation
22. Unconsciousness
23. Any Other (.....)
24. **Multiple Indication -** Mention All Relevant Numbers: a ..... b ..... c ..... d.....

Provisional Diagnosis :

***Final Diagnosis** (Encircle the most relevant single diagnosis, If multiple causes also mention all relevant numbers in the end as per sequence)

- ELB (999 gm or less) : P 07.0
- Other LBW (1000 gm - 2499 gm) : P 07.1
- Extreme Immaturity (<28 Weeks) : P 07.2
- Prematurity (28-<37 Weeks) : P 07.3
- Small for Gestational Age (IUGR) : P 05.1
- Neonatal Aspiration of Meconium : P 24.0
- RDS of Newborn (HMD) : P 22.0
- Transient Tachypnoea of Newborn : P 22.1
- Pneumothorax : P 25.1
- Congenital Pneumonia : P 23
- Acquired Pneumonia : J 15
- Primary Sleep Apnoea of Newborn : P 28.3
- Birth Asphyxia : P 21.0
- HIE of Newborn : P 91.6
- Neonatal Sepsis : P 36.9
- Meningitis : G 00
- Convulsion of Newborn : P 90 (Hypoxic, Hypoglycaemic, Hypocalcaemic, CNS Infections, Birth Trauma, Metabolic, Other, Unknown Cause)
- Hemolytic disease of Newborn : P 55
- Neonatal Jaundice : P 59
- Acute Renal Failure : N 17
- Neonatal Cardiac Failure : P 29.0
- Shock : R 57
- DIC : P 60
- Intraventricular Hemorrhage : P 52.3
- Neonatal Diarrhoea : A 09
- Tetanus Neonatorum : A 33
- Hypothermia of Newborn : P 80
- Environmental Hyperthermia of Newborn : P 81.0
- Neonatal Hypoglycaemia : P 70.4

***(Based on WHO, ICD - 10 Version : 2010)**

- Congenital Malformation :
  (a) Cong. Diaphragmatic Hernia : Q 79.0
  (b) Cong. Hydrocephalus : Q 03
  (c) Meningomyelocele : Q 05
  (d) Imperforate anus : Q 42.3
  (e) T.O. Fistula : Q 39.2
  (f) Congenital Heart Disease : Q 21
  (g) Cleft Palate : Q 35
  (h) Cleft Lip : Q 36
  (I) Cleft Palate with Cleft Lip : Q 37
  (j) Congenital Deformities of Hip : Q 65
  (k) Congenital Deformities of Feet Q 66
  (l) Other Malformation (.....)
- Any Other Diagnosis (.....)
- **Multiple Diagnosis**-Mention All Relevant Codes : a ..... b ..... c ..... d .....

This Sheet has to be filled on Admission by Doctor on Duty

MOTHER'S INFORMATION : Past History and ANC Period

Mother's Age ..... Yrs. Mother's Wt. ..... Kgs. Age at Marriage ..... Yrs.

Consanguinity : Yes [ ] No [ ] Birth Spacing : <1 Yr / 1-2 Yr / >2-3 Yr / > 3 Yr / Not Applicatble

Gravida : ..... Para : ..... Live Birth : ..... Abortion .....

LMP : ..... / ..... EDD : ..... / ..... / ..... Gestation Weeks : .....

Antenatal Visit's : Non / 1 / 2 / 3 / 4 T.T.Doses : None / 1 / 2

Hb : ..... Blood Group : .....

PIH : No [ ] Yes [Hypertension / Pre Eclampsia / Eclampsia]

Drug : No [ ] Yes [ ] (.....) Radiation : Yes [ ] No [ ]

Illness : Malaria / T.B. / Jaundice / Rash with Fever / U.T.I. / Syphilis / Other (.....)

APH : Yes [ ] No [ ] GDM : Yes [ ] No [ ]

Thyroid : Euthyroid (N) / Hypothyroid / Hyperthyroid / Not Known

VDRL : Not Done / +Ve / -Ve HbsAg : Not Done / +Ve / -Ve

HIV Testing : Done / Not Done Amniotic Fluid Volume : Adequate / Polyhydraminos / Oligohyd.

Other Signification Information :

MOTHER'S INFORMATION : During Labour

Antenatal Steroids : Yes [ ] No [ ] If Yes, Betamethasone [ ] / Dexamethasone [ ]

No. of doses : [1] [2] [3] [4] Time Between Last Dose & Delivery ..... hrs./..... Days

H/O Fever : In 1st Trimester / In 2nd Trimester / In 3rd Trimester / During Labor only if > 100.4 F

Foul Smelling Discharge : Yes [ ] No [ ] Uterine Tenderness : Yes [ ] No [ ]

Leaking P.V. > 24 Hours : Yes [ ] No [ ] PIH : Hypertension / Pre Eclampsia / Eclampsia

PPH : Yes [ ] No [ ]

Amniotic Fluid : Clear / Blood Stained / Meconium Stained / Foul Smelling

Presentation : Vertex / Breech / Transverse Labour: Spontaneous / Induced

Course of Labour : Uneventful / Prolonged 1st stage / Prolonged 2nd stage / Obstructed

E/O Feotal Distress : Yes [ ] No [ ] Type of Delivery : LSCS / AVD / NVD

Indication for Caesarean, if Applicable : [Cephalo Pelvic Disproportion] [Malpresentation] [Placenta Previa] [Obstructed Labor] {Foetal Distress] [Prolonged Labour] [Cord Prolapse] [Failed Induction (Dystocia)] [Previous LSCS] [Other.....]

Delivery Attended by : [Doctor] [Nurse] (ANM) [Dai] [Relative] [Any Other] .....

Other Significant Information :

If Information is Not Available, Leave the Field Blank, Do Not ✓ "No [ ]"

BABY'S INFORMATION : At Birth

Cried Immed. after Birth	:	Yes [ ] No [ ]	Wt. at Birth : ..... Kgs.
Gestational age	:	..... in completed weeks	Maturity : Preterm (<37 Wk) / Full term / Post term (>42 Wk)
APGAR at 1 Min	:	..... / Not Available	APGAR at 5 Min : ..... / Not Available
Resuscitation Required	:	NO [ ] Yes [ ] Tactile Stimulation / Only Oxygen / Bag & Mask [Duration ..... min.] / Intubation / Chest Compression / Adrenaline	
Vitamin K Given	:	Yes [ ] No [ ]	Breast Fed with 1 Hour : Yes [ ] No [ ]

BABY'S INFORMATION : On Admission

PRESENTING COMPLAINTS :

GENERAL EXAMINATION

General Condition	: [Alert] [Lethargic] [Comatose]	Temperature ..... ^{0}C	Heart Rate...../min
Apnea	: Yes [ ] No [ ]	RR ..... / min.	B.P. : .....
Grunting	: Yes [ ] No [ ]	Chest Indrawing : Yes [ ] No [ ]	
Head Circumference	: .....c.m.	Length ..... c.m	
Color	: Pink / Pale / Central Cyanosis / Peripheral Cyanosis		
CRT >3 secs	: Yes [ ] No [ ]	Skin pinch > 2 secs : Yes [ ] No [ ]	
Meconium Stained Cord	: Yes [ ] No [ ]	Cry : Absent / Feeble / Normal / High Pitch	
Tone	: Limp / Active / Increase Tone	Convulsions : Present on Admission / Past History / No	
Jaundice	: Yes [ ] No [ ] If Yes, extent [Face] [Chest] [Abdomen] [Legs] [Palms / Soles]		
Bleeding	: Yes [ ] No [ ] If Yes, specify site [Skin] [Mouth] [Rectal] [Umbilicus]		
Bulging Anterior Fontanel	: Yes [ ] No [ ]	Taking Breast Feeds : Yes [ ] No [ ]	
Sucking	: [Good] [Poor] [No Sucking]	Attachment : [Well attached] [Poorly attached] [Not attached]	
Umbilicus	: [Red] [Discharge] [Normal]	Skin Pustules : [No] [Yes <10] [Yes >=10] [Abscess]	
Congenital Malformation	: No [ ] Yes [ ] Diaphragmatic Hernia / Hydrocephalus / M.M.C./ Imperforate Anus / T.O. Fistula / Cong. Heart Disease / Cleft Palate / Cleft Lip / Cleft Palate with Cleft Lip / Cong. Deformity of Hip / Cong. Deformity of Feet / Other		
Blood Sugar	:	Oxygen Saturation :	
Other Signification Information:			

If Information is Not Available, Leave the Field Blank, Do Not ✓ "No [ ]"

SYSTEMIC EXAMINATION

CVS : .....

RESPIRATORY : .....

PER ABDOMEN : .....

CNS : .....

OTHER SIGNIFICANT FINDING : .....

TREATMENT ADVISED : On Admission

INVESTIGATIONS ADVISED : On Admission

Foot Print of Newborn
(Left Foot)

Doctor's Name and Signature

Date : **Signature :**

Time : **Name** **Relationship :** **Sign.: Doctor / Nurse**

FINAL OUTCOME

Successfully Discharged / Left Against Medical Advice / Referred / Expired

In Case of Death : Mention Cause of Death (✓ The Most Relevant Single Indication)

1. Respiratory Distress Syndrome
2. Meconium Aspiration Syndrome
3. HIE / Moderate - Severe Birth Asphyxia
4. Sepsis
5. Pneumonia
6. Meningitis
7. Major Congenital Malformation
8. E.L.B.W. (Wt. less than 1000 g)
9. Prematurity (<28 weeks of Gestation)
10. Neonatal Tetanus
11. Cause not established
12. Any Other : .....

.....

.....

This Sheet has to be filled on Admission by Doctor on Duty

தகவல் படிவம்

மதிப்பிற்குடிய ஐயா / அம்மையீர்

உங்கள் விருப்பதின் பேரில் இரத்தத்தில் கிருமி தொற்றுதல் என சந்தேதிக்கும் பச்சிளங்குழந்தைகளின் இரத்தத்தில் சி **ரியாக்டிவ் புரோட்டின்** (சி.ர.பி) இரத்த அணுக்களின் எண்ணிக்கையை மதிப்பிடுவதன் மூலம் கிருமி தொற்றுதலை விரைவாக கண்டறிவதற்கான ஆய்வில் உங்கள் குழந்தையை உட்படுத்துவீர் என அன்புடன் கேட்டுக் கொள்கிறோம். இந்த ஆய்வில் ஆராய்ச்சி நோக்கத்திற்காக உங்கள் குழந்தை பரிசோதனைக்கு உட்படுத்தப்பட்டு தகுந்த சிகிச்சை விரைவாக அளிக்கப்படும். தங்களுக்கு உங்கள் குழந்தையை இந்த ஆய்வில் உட்படுத்த விருப்பம் இருந்தால் தாங்கள் அருள்கூர்ந்து ஒப்புதல் படிவத்தைப் படித்துப் பார்த்து கையொப்பம் இடும்படி கேட்டுக் கொள்கிறேன்.

ஆய்வாளரின் பெயர் **மரு. வே. கோமதி**

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

கிருமிதொற்றுதலால் பாதிக்கப்பட்டுள்ள பச்சிளாங்குழந்தைகளின் இரத்தத்தில்,சி – ரியாக்டிவ் புரோட்டின் (சி.ர.பி) அளவினை மதிப்பிட்டு அதனை இரத்த அணுக்களின் எண்ணிக்கையோடும் மருத்துவ அளவுருகளோடும் ஒப்பிடுவதற்கான ஆய்வு

ஆராய்ச்சி நிலையம் – அரசு ஸ்டான்லி மருத்துவமனையின் ஒரு அங்கமான அரசு இர.எஸ்.ர.எம், மருத்துவமனை, சென்னை.

ஆய்வில் ஈடுபடுத்தப்படும். குழந்தையின் பெயர் ..... (அ) பெற்றோரின் பெயர் ..... (தாய் / தந்தை / காப்பாளர்) ஆய்வில் ஈடுபடுத்தப்படும் குழந்தையின் எண் .....

மேலே குறிப்பிடப்படுள்ள ஆய்வின் விவரங்கள் எனக்குவிளக்கப்பட்டது.

என்னுடைய சந்தேகங்களைக் கேட்கவும், அதற்க்கான தகுந்த விளக்கங்களைப் பெறவும் வாய்ப்பளிக்கப்பட்டது. நான் இவ்வாய்வில் மருத்துவமனையில் அனுமதிக்கப்பட்டுள்ள என்னுடைய குழந்தையை தன்னிச்சையாகத் தான் ஈடுபடுத்தியுள்ளேன். ☐

எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்டச்சிக்கலுக்கும் உட்படமால் நான் இவ்வாய்வில் இருந்து எனது குழந்தையை விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ இதைச்சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும்போதும் இந்த ஆய்வில் பங்கு பெறும் மருத்துவர் என்னுடைய குழந்தையின் மருத்துவ அறிக்கையை பார்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து எனது குழந்தையை விலகிக்கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வுமூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்க்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதைப்பிரசுரிக்கவும் என் முழுமனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் மருத்துவமனையின் அனுமதிக்கப்பட்டுள்ள எனது குழந்தையை ஈடுபடுத்த ஒப்புக்கொள்கிறேன் ☐

பங்குபெறும் குழந்தையின் பெற்றோர் (தாய் (அ) தந்தை) காப்பாளரின் கையொப்பம் .....கட்டை விரல் ரேகை.....
இடம் .....தேதி..... குழந்தையின் பெற்றோர் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி .....

ஆய்வாளரின் பெயர் **மரு. வே. கோமதி**